THE EFFECT OF TISSUE EXPANSION ON THE CIRCULATION OF SKIN

Thesis presented for the degree of ChM

Edinburgh University

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1990

DECLARATION

This thesis has been composed by myself and the experimental work described within has also been conducted by myself.

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ABSTRACT

Although the generation of skin flaps by controlled hydraulic distension, known as Tissue Expansion, is a relatively new technique in Plastic Surgery considerable clinical experience has been gained in this field.

In order to raise skin flaps safely without necrosis it is necessary to understand their vascular anatomy. Both the insertion of and the inflation of tissue expanders causes changes in the blood supply of the overlying skin and thus the planning of skin flaps in expanded skin is subject to extra considerations in addition to those governing conventional skin flaps. Although it has been suggested that tissue expansion promotes an increase in skin vascularity there has been a lack of scientific investigation into the pathophysiological changes that occur during this procedure.

It has been the purpose of this study to determine what changes occur in the circulation of skin flaps when they are subjected to tissue expansion. The pig buttock flap has been studied for its suitability as an experimental model. By placing expanders under each buttock and by expanding one side only, leaving the other side empty as a control, it was possible to investigate

changes due to expansion only, in isolation of any effect caused by the creation of a subcutaneous pocket or insertion of the prosthesis.

In the first experiment the effect of tissue expansion on the territory of cutaneous blood vessels has been investigated by determining the survival of the buttock flap after it has been raised as an island. An electro-magnetic flowmeter was used in the second experiment to study blood flow in the axial vessel vessel of the flap. In the final part of the study the structure of the vessels was investigated by angiography, histological analysis and direct measurement of the axial vessel calibre.

The area of skin which could be sustained by a specific cutaneous vessel was found to be significantly increased following tissue expansion, apparently directly proportional to the degree of expansion. However contrary to expectations this increase in area was not accompanied by any increase in blood flow through the vessel concerned. Some increase in the number and cross-sectional area of the small blood vessels in the skin were found and in particular a plexus of small vessels appeared to develop at the interface of the capsule which forms around the implant and the rest of the flap. This was not matched by an increase in number or size of the larger vessels which elongate in response to the expansile forces partly by uncoiling.

In conclusion the expected increase in vascularity as a result of tissue expansion was not found and in reality there may be some stagnation of blood within an enlarged vascular compartment. The effect of these findings on design of flaps within expanded skin are discussed.

ACKNOWLEDGEMENTS

I wish express my gratitude to the following who have assisted me in this work.

Mr R.A.W.McDowall MChir, FRCS, Consultant Plastic Surgeon, Odstock Hospital,Salisbury.

Mr D Cranstone, Director, Cory Brothers.
Cox Uphoff.

Mr T Richards, Senior Animal Technician, Southampton General Hospital.

Wessex Regional Health Authority.

Ministry of Defence, Porton Down, Wiltshire.

Professor W.H.Reid FRCS, Consultant Plastic Surgeon, Canniesburn Hospital, Glasgow.

Peel Medical Trust.

Professor M Harper, Steward, Wellcome Surgical Institute.

Miss C.Stirton, Senior Animal Nurse, Wellcome Surgical Institute.

Mr J White, Dow Corning Ltd.

Professor K. Simpson FRCPath, Department of Pathology, Glasgow Royal Infirmary.

Dr. J. Vance, Department of Anaesthetics, Glasgow Royal Infirmary.

Mr G Littlejohn, Senior technician,Wellcome Surgical Institute.

Mr D Soutar FRCS, Consultant Plastic Surgeon, Canniesburn Hospital.

Mr C M Ward FRCS, Consultant Plastic Surgeon, Charing Cross Hospital.

Mr M R Masser FRCS, Senior Registrar in Plastic Surgery, Odstock Hospital.

Mr. C.V.Ruckley, ChM, Consultant Vascular Surgeon, Edinburgh Royal Infirmary.

Dr. M. Barrat, Department of Histopathology, Charing Cross Hospital.

Mr J.J. McCann, Consultant Plastic Surgeon, University Hospital, Galway.

Mr D.M.Davies, Consultant Plastic Surgeon, Charing Cross Hospital, London.

Miss Linda Thursby, Medical Artist, Charing Cross Hospital.

Mr R. Barnett, Medical Photographer, Charing Cross Hospital.

Medical Photography Department, Canniesburn Hospital, Glasgow.

Finally I would like to express my gratitude to my wife, Judith, for her patience and support throughout this work.

CONTENTS

CHAPTER 1 INTRODUCTION

One of the fundamental methods by which the plastic surgeon manages to transfer tissue for reconstruction is by using skin flaps. As defined by Grabb (1979) "A skin flap consists of skin and subcutaneous tissue that is moved from one part of the body to another with a vascular pedicle or attachment to the body being maintained for nourishment". It is thus essential that the surgeon has a full knowledge of the anatomy of the blood supply of the skin in which he plans to design and raise a skin flap.

Blood flow through skin depends little on the nutritional requirements of the skin but on the requirements of body thermoregulation. At room temperature skin blood flow in man is 10 - 15 ml/min/100 gm.tissue and this is many times that required for perfusion of the skin itself, (Keele et al 1982). The situation is dramatically changed in skin when it is raised as a skin flap. As blood vessels which cross the margins of the flap are divided there is a reduction in the blood flow of the flap which is most marked in the first twelve hours, (Tuschida 1976 & 1978, Sasaki and Pang 1980). If the flap is badly designed the blood flow to the periphery of the flap may drop below a level critical for flap survival with subsequent failure and necrosis of the flap.

As there was an increase in understanding of how

skin derived its blood supply there was a concomitant evolution in the type of skin flap that became available for the reconstructive surgeon. Initially it was not appreciated that there are certain cutaneous blood vessels which run specific courses in the subcutaneous tissues and the first generation of skin flaps were designed without taking these vessels into account. Because of the limited vascularity of such flaps, known as Random Pattern Flaps, it is generally recommended that the length to breadth ratio of these flaps is limited to 1:1 to reduce the incidence of avascular necrosis of the distal end of the flap. Despite these constraints random pattern flaps carry an appreciable risk of flap failure in clinical practice due to inadequacy of blood supply.

As the cutaneous circulation was better understood flaps were designed to include a direct cutaneous artery and its accompanying venae commitantes, known as Axial Pattern Flaps. Consequently it was possible to design flaps at least as long as the axial vessel regardless of flap width.

In axial flaps with a large enough axial vessels the flap may be refined by removing all other attachments except the vessels alone producing what is known as an Island Flap.

A further degree of sophistication was made possible with the advent of Microvascular Surgery which permitted an island flap to be completely detached from the body and reattached at a distant site by anastomosing the axial vessels with suitable vessels at the recipient

site.

Further generations of flaps have evolved with the discovery of other sources of blood supply to the skin, principally from underlying deep fascia and muscle. Further description of the design of skin flaps are beyond the scope of this work and the reader is referred to Mcgregor (1980), Converse et al (1977) and Grabb (1979).

Conventional skin flaps are taken from a donor site which may itself require to be repaired if there is insufficient redundant tissue locally. However with the new technique known as tissue expansion an increased area of skin for reconstruction can be made available which may be sufficient to cover both donor site and defect. It thus becomes possible to use skin immediately adjacent to a defect which will produce skin of excellent colour and texture match, (Manders et al 1984). In this technique the skin to be expanded is first prepared by a preliminary operation in which a subcutaneous pocket is created into which a tissue expander is inserted. This consists of a silicone elastomer balloon which can be inflated through an attached valve. As soon as the wound is considered to be strong enough, the balloon is inflated by serial injections of normal saline and progressive distension of the overlying skin occurs with an increase in its area. When the area of skin is judged to be sufficient for the intended reconstruction a further operation is performed in which the prosthesis is

removed and the expanded skin used to effect the reconstruction.

Considerable clinical experience has been gained in tissue expansion but despite this a very high complication rate is common, quoted as high as 40% in some series, (Dickson and Sharpe 1987 and Antonyshyn et al 1988.). It is likely that a new technique would rapidly fall into disrepute in the presence of such a high complication rate and this would presumably have happened to Tissue Expansion had it not been for the excellent cosmetic results that can be achieved with this method, (Fenton 1988.).

The high complication rate may, in part, be related to a comparative lack of detailed scientific research into the pathophysiology that occurs as a result of Tissue Expansion. In no area is this more important than the blood supply. In an early description of tissue expansion Radovan described the erythema that often occurs during expansion and attributed it to an increase in vascularity in the expanded skin. Although this phenomenon has also been alluded to by several others, (Cherry 1983, Antonyshyn 1984), there is a lack of scientific evidence to support this hypothesis.

There are many external factors which affect skin blood flow. The most major specific one is the sympathetic tone which effects thermoregulation, (Keele et al 1982). Other interrelated factors are cardiac output, blood pressure and blood viscosity. If all these

systemic variables are excluded the principle factor controlling the perfusion of a flap is its local vascular anatomy and this includes both the topographical anatomy of the vessels and their size and structure.

Important changes occur in the vasculature of the skin when a subcutaneous pocket is created at the time of insertion of a tissue expander and during the process of expansion itself. Thus the planning of skin flaps in expanded skin is subject to extra considerations in addition to those governing conventional skin flaps.

In order to determine the limits within which a flap designed in expanded skin can safely be raised the following questions are to be answered in this study:

1. **Does the area of skin supplied by a specific cutaneous vessel increase after expansion?**

2. **Is the blood flow through cutaneous vessels increased?**

3. **Does the weight of tissue supplied by a specific vessel increase and hence affect the perfusion per unit weight of tissue?**

4. **What is the effect of expansion on the structure of the vascular tree, both macroscopically concerning vessel topology and microscopically concerning perfusion of tissues and suitability of vessels for microvascular anastomoses?**

CHAPTER 2 CLINICAL APPLICATION OF TISSUE EXPANSION

2.1. DEVELOPMENT OF TISSUE EXPANSION

The elongation of tissues by distraction is not a phenomenon new to man. It is a custom that has been practised by certain races for a long time for cosmetic purposes. In Padaung, Burma, long slender necks are considered attractive and members of the tribe fit wire collars around the neck to which are steadily added others with the result that there is elongation of the neck by gentle traction on all of its tissues, (Burnet W 1945).

A similar example occurs in certain tribes in Chad where projecting lower lips are favoured. Tribesmen fit a disc into the lower labial sulcus which is replaced by discs of sequentially increasing diameter until the lip is large enough to accommodate a disc the size of a tea-plate, (Weeks G.S. 1956), (Figure 2.1).

Although all connective tissues of the body, including bone (Matev 1980), may be elongated by prolonged stretching, it is the production of an increased area of skin that has the greatest application in reconstructive plastic surgery. The capacity of skin to stretch under forces of gradual distraction to an almost unlimited degree and its application to wound closure was first described in the early part of this century, (Morestin, 1915 and Staige Davis 1929). These surgeons describe the removal of large cutaneous

lesions, too big for total excision and primary closure, by the excision of as large a part of the lesion as can be closed by direct suture. After the wound is well healed and the surrounding tissues have regained their elasticity, after a period of two to three months, a further resection is made and this procedure is repeated until the lesion has been completely removed. If used correctly this technique, known as serial excision, allows the removal of large lesions without skin closure under unacceptable tension thus avoiding problems of distortion such as ectropion, (Wilson,J.S.P. 1949). In this form of reconstruction the elongation of skin takes place immediately after wound closure and it took a further 40 years before the generation of skin prior to reconstruction was achieved surgically.

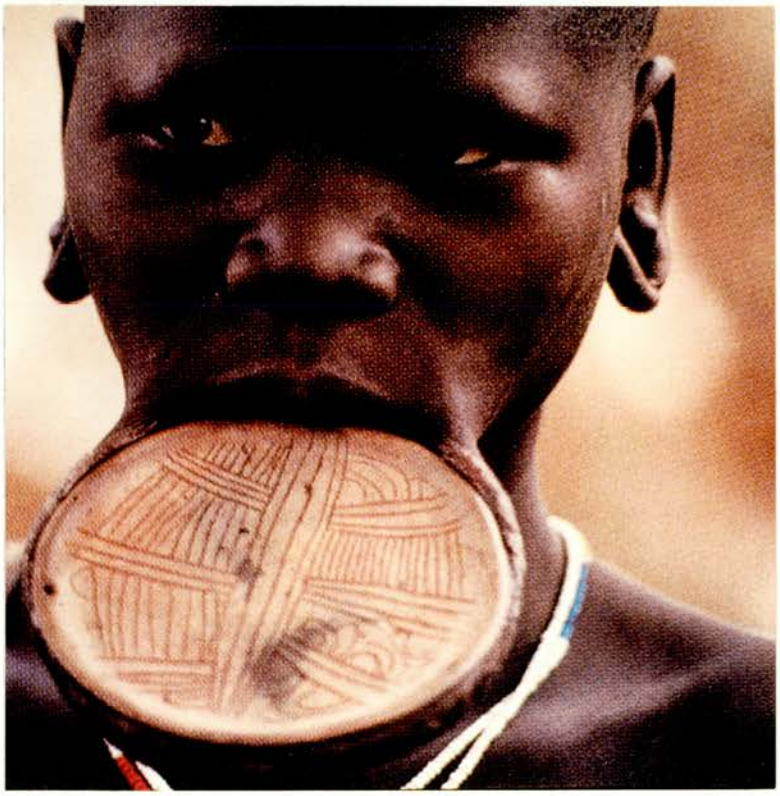


Figure 2.1 Lip stretching practised by Chad tribesman

It is common knowledge that skin will increase in area to accommodate a growing underlying mass; the pregnant abdomen being the example frequently quoted. Pregnancy is however a comparatively gradual process occurring over a period of nine months; much more dramatic growth of skin is seen over rapidly growing neoplasms and haematomata which may cause a dramatic increase in the area of overlying skin.

This phenomenon is utilised in Tissue Expansion which is the term given to the technique of increasing skin surface area by controlled hydraulic distension of a surgically implanted subcutaneous device. It is generally accepted that this technique was first employed for the purpose of surgical reconstruction by Neumann, (1956). His case involved a male patient aged 52 who had suffered traumatic loss of the upper pole of his right ear 15 years before reconstruction. A rubber balloon 5 inches long by 1 inch in diametre was placed subcutaneously immediately superior to the external auditory meatus and connected to the external environment by a polythene tube. Over a period of two months air was injected several times a week until the area of the overlying skin had increased by 50%. The balloon was then removed and the skin flap created used to cover an autogenous cartilage graft, thus reconstituting the upper pole of the ear. Following this case reported in 1957 little interest was shown in this technique until it was rediscovered twenty years later.

The late Dr. C. Radovan is credited with popularising this technique and in 1976 became the first surgeon to expand a skin flap in order to resurface an adjacent defect. From his initial design of a temporary expander have been developed the expanders in clinical use today. The device consists of a balloon made of a silicone elastomer connected by tubing to a valve, (Figure 2.2). This valve is filled with a silicone gel and permits saline to be injected into the system with a small hypodermic needle thus inflating the balloon. Inflation causes a distending force to be exerted on any tissue that overlies it. Although many refinements have been made to all parts of this system the basic design remains unchanged in current expanders.

Figure 2.2 Tissue Expander showing balloon, valve and connecting tubing.

Devices have been developed with the valve incorporated in the wall of the balloon with no connecting tube. Although these are simpler to insert because no tunnel needs to be dissected for connecting tube and valve they have the major disadvantage that the valve is more difficult to locate and inaccuracy in injection may result in perforation of the balloon, (Muenker 1988).

Shortly after Radovan's initial trials another group independently started working in this field in this field on a self inflating tissue expander, (Austad 1982). This device also consisted of a balloon made from a silicone elastomer but unlike the Radovan expander it had no valve and sealed inside the device was a solute load of sodium chloride. When the device was implanted subcutaneously fluid was drawn in from the surrounding extracellular fluid compartment under osmotic pressure. Over a period of weeks the tissue expander inflated until the fluid contained within became isotonic with that surrounding it. This device was used successfully in at least two patients but further testing in animals led to concern about implant rupture and tissue necrosis from the resulting extravasation of hypertonic saline and thus further clinical trials have been postponed.

In the early cases performed by Radovan and Austad the defects treated were areas of skin deficiency. An area of adjacent skin was expanded producing a skin flap large enough to cover both the defect and the donor site.

In subsequent cases the technique has also been used where there is not only a skin shortage but absence of an underlying structure, as that described in Neumann's original case. Here the skin of the defect itself is expanded, restoring skin area and allowing the placement of a permanent implant into the expander cavity. In this way missing three dimensional structures such as the breast can be reconstructed, (Lapin 1980).

In the current practice of tissue expansion an initial operation is performed in which a subcutaneous pocket is created for the placement of the implant. There is a difference of opinion concerning the best site for the incision for placement of the expander, (Radovan 1984, Argenta 1984, Manders 1986). If a defect is to be excised the simplest site for the incision is along the margin of the defect. This will not prejudice the final design of flaps to be raised in the expanded skin and the scar may be excised along with the defect. However subsequent tissue expansion will exert tension across the wound possibly increasing the risk of wound dehiscance and implant exposure. Although Radovan suggested that incisions should not be placed across the advancing edge of tissue but in a radial direction as an expander has to obey Newton's laws it will exert a distending force in all directions and any incision over the expander will be subject to distracting forces. The only way to avoid this problem is to place the incision at some distance from the skin to be expanded and a subcutaneous dissection made to the proposed site of the expander.

Normally the device is completely buried subcutaneously although experience recently reported, (Jackson 1987 and Dickson 1988), suggests that it is possible to have the connecting tubing passing through a small wound in the skin leaving the valve external. This has the obvious benefit that no skin puncture is necessary for inflation of the expander which makes the serial injections less frightening for children. However it is doubtful whether, if an external valve is used, the expander cavity will remain sterile which will preclude the use of this technique if the expander is to be replaced by a long term prosthesis, e.g. for breast reconstruction.

After the elapse of a period of time for wound healing to occur the expander is serially inflated. The rate of inflation is partly controlled by the elasticity of the skin and partly by convenience to both patient and surgeon. Rigorous aseptic technique is observed for the injection of fluid. The valve has to be punctured with a small 23 gauge needle to inject saline, as larger needles will allow leakage of saline back out of the puncture holes in the valve. Some surgeons prefer to monitor pressure within the lumen of the system to prevent the risk of overinflation, (Hallock and Rice 1986), and recommend that inflation ceases when a pressure of 40mms. Hg. has been reached. Because injection is almost certain to produce pain before this pressure is reached and the expander will feel tense, in practice objective pressure monitoring is not often employed. Depending on the skin

to be expanded the firmness produced by inflation will soften from between several hours to several days following injection, thus permitting further inflation.

When it is estimated that a large enough increase in skin surface area has been obtained a further operation is performed in which the expander is removed and the skin utilised for reconstruction.

If the expander is intended to permit a three dimensional reconstruction in the same site a small incision will be required to remove the expander and replace it with a permanent structure, for example a breast prosthesis in a breast reconstruction or an ear skeleton in an ear reconstruction. Alternatively if the expander is being used to resurface an adjacent defect the surgeon must decide how best to use the skin available for reconstruction. The flap design must not only maintain skin vascularity but allow the flap to be transferred from the convex dome of the expander into the defect. Direct advancement of the flap by an incision along the edge of the expander adjacent to the defect is the simplest design to execute but as the skin around the margin of the prosthesis has not been expanded it will limit linear advancement of the maximum area of skin availability which is over the dome of the expander.

There is a place for advancement flaps in scalp reconstruction when the curvature of the inflated expander matches that of the part of the skull to be resurfaced but in other sites better use of the skin may

be achieved by raising a transposition flap from the expanded skin to cover the defect leaving sufficient skin behind to cover the donor defect. In this manner maximum utilisation of the skin may be achieved with the minimum of scarring.

Much recent work to improve expander design has been aimed at making transfer of skin from expander to defect more simple. With the help of mathematicians and engineers computers have been asked to design an expander shape which will generate skin in such a manner that defects may be closed by simple advancement. For a round defect a crescentic or croissant shape is recommended, (Fenton 1987). The expander is implanted with the lesion to be excised lying in the concavity of the expander. If the lesion is then excised as an ellipse in the same longitudinal axis as the expander the skin may easily be advanced across the resulting defect. The resulting scar will be considerably shorter than that from advancement using a round expander. For other shaped defects different designs of expander may be appropriate, (Brobman and Huber 1985).

2.2. CLINICAL PRACTICE

2.2.1. HEAD AND NECK RECONSTRUCTION

In no other site is an acceptable cosmetic result more important than in the head and neck. Conventional reconstruction, however sophisticated, using tissue from distant parts of the body whether as skin flaps or skin grafts often fails to completely match the surrounding tissues. When skin is transported to another site it cannot change its characteristics and will always resemble skin of the donor site. By enabling the use of local skin tissue expansion can produce a result in which the reconstructed area is likely to blend in much more with its surroundings than hitherto possible. For this reason it is now possible to correct facial deformities which in the past could not have been improved. An important factor if this technique is used in the head and neck is that there is likely to be considerable disfigurement temporarily whilst tissue expansion is completed. It is therefore important that patients are selected carefully and that they fully understand what is involved. Patients who are psychologically disturbed are unlikely to tolerate the treatment although well adjusted patients may well continue a normal life including their regular employment. Experience has been gained with many different areas in the head and neck and these will briefly be discussed.

Scalp

The term scalp reconstruction is used imprecisely to describe two different objectives. Firstly it is often used to describe the skin cover of an exposed wound over the cranium. This may involve transfer of skin grafts, skin flap or muscle flap as the situation dictates. The primary objective is to obtain skin cover regardless of hair. In the acute injury this is still normally achieved by conventional means. The second usage of the term scalp reconstruction is to describe the restoration of hairbearing skin in a part of the scalp which has become bald. Common reasons for this are burns scarring, alopecia and male pattern baldness. In the past this has been achieved in two ways. Small "punch grafts" of skin have been transferred from hairbearing skin, usually in the occipital region, and transferred to the area in need, (Ayres 1985). Although some patients are well pleased by this procedure, on close inspection the tufts of transplanted hair can easily be identified and give the reconstruction a "toothbrush" appearance. The other method of hair transfer involves the reconstruction of an anterior hair line by transposition of bilateral temporal scalp flaps, (Jure 1975). To obtain direct closure of the donor site these flaps must be of limited width which means that, although they may be used in cases where there is a large area of alopecia, they will not permit all of the hairless skin to be removed and must therefore be used to disguise the persisting areas of hairloss.

Tissue expansion has added a new dimension to scalp reconstruction. If sufficient hairbearing skin remains it is possible to dramatically increase its area and resurface large bald areas. Of course the sum total of hair follicles is not increased and so what in effect happens is a redistribution of the existing hair follicles although in a much more even manner than could be obtained by punch grafts. The appearance of hair density is a product of hair shaft crossectional area and hair follicle density. It has been estimated that the hair will not look unduly thin provided that a hair:skin ratio of more than 1 mm^2 of cross sectional area of hair to 1 cm^2 area of scalp is maintained, (Masser 1988). As the ratio is normally double this figure in the normal scalp and even higher in the thicker occipital regions it is possible to remove large areas of alopecia without unduly thinning the hair, (Manders et al 1987). It becomes progressively more important the bigger the area to be resurfaced that there is a fairly clear demarcation between healthy hairy skin and the bald areas. An area of patchy alopecia remains difficult to reconstruct without sacrificing all tufts of hair which may be present.

After making the appropriate incisions a sub-galeal dissection is made. This is usually done quickly and easily by blunt dissection. It is important that more than one expander is used if the area to be reconstructed is large and the best site for expansion, if adjacent to

the defect, is the occipital region due to the higher hair follicle density. Once the expander has been inserted and the wound closed there is usually enough elasticity in the flap to permit inflation of some saline intra-operatively. This has the benefit not only of filling out the wrinkles within the device but also of reducing the dead space in the subcutaneous pocket and reducing the risk of haematoma formation.

The first 2 to 3 weeks of scalp expansion is likely to be slow and possibly uncomfortable. The resistance of the Galea is then overcome and larger amounts of saline can be injected with an acceleration in the gain of surface area,(Argenta 1984).

The geometry of flap transfer of the expanded skin is beyond the scope of this work and the reader is referred to more detailed accounts, (Leonard and Small 1986 and Manders et al 1984).

Case Report 1

A 12 year old girl sustained loss of an area of scalp approximately 11 cm. by 7 cm. in a road traffic accident. This was immediately repaired with a split skin graft which healed leaving an obvious area of alopecia which she wanted to be removed, (Figure 2.3). Twin expanders were inserted, one on either side of the defect, and over a period of 7 weeks were inflated to a volume of 300 ml. each, (Figure 2.4). At a further operation the expanders were removed and the area of alopecia was completely excised, (Figure2.5).

Figure 2.3 Following the initial repair of a traumatic wound there is a large area of alopecia.

Figure 2.4 The scalp with twin fully inflated expanders before definitive repair

Figure 2.5 Scalp after completion of reconstruction

Forehead

The forehead has been successfully expanded both for local use to resurface adjacent forehead defects and for use as a distant pedicled flap. The expander may be placed under the skin alone or under the frontalis muscle. Submuscular placement provides the opportunity to create a flap which is not only sensate but includes functioning muscle which will produce the best repair of local defects. However submuscular expansion of the forehead is often found to be a painful procedure and it has been suggested that this is due to ischaemia of the muscle on inflation,(Gault et al 1988).

Considerable experience has been gained in the field of reconstructive rhinoplasty using expanded forehead

skin. Besides the obvious advantage of primary wound closure of the forehead donor defect there are also benefits to the flap itself which becomes much thinner than would be raised without prior expansion. As a result when the skin is draped over the nasal skeleton more definition will be achieved in the reconstruction, (Adamson 1988). It is important that a skeletal support is provided for the expanded skin because without one there will be a tendency for contraction of the skin to occur with loss of shape of the nose tip. Some workers have found this a problem, (Bolton et al 1988), and would not now advocate pre-expanding the forehead flap but, after completing the reconstruction in conventional fashion with a graft to the donor site, would expand the remaining forehead to enable subsequent removal of the graft.

Neck and Cheek

The neck and cheek are considered as a single anatomic unit for purpose of tissue expansion as the skin of both is of similar thickness and hairbearing properties. Expanded skin can thus be transferred up from the neck to the cheek producing a good match, (Antonyshyn et al 1988).

The usual lesions in the cheek that are suitable for reconstruction by this technique are haemangiomata, naevi and areas of burns scarring. If the cheek is to be expanded the device is normally inserted through a face

lift incision to minimise scarring and placed in a subcutaneous position superficial to the facial muscles to avoid damage to the facial nerve, (Argenta et al 1983). For larger defects it may be necessary to import skin from the neck. Most authors recommend placing the expander beneath the platysma in the neck but it has been suggested that having the extra bulk of the platysma in the flap will obscure the cervico-mandibular angle, (Marks et al 1987). In transferring skin from the neck to the face in order to maintain this angle it is much more important to ensure that a sufficient amount of skin has been generated to allow it to drape into the hollow of the neck without tenting accross, if necessary with a few sutures hitching the dermis of the flap up to the deeper tissues in the hyoid region.

A frequent reason for neck reconstruction is for the correction of burns contractures which are a source of major functional and cosmetic disability.

For the purpose of planning these contractures can be divided into two groups; central contractures sparing hairless skin on either side of the neck and lateral contractures sparing skin only on one side, (Saxby and Gowar 1988). The former can be resurfaced by bilateral expanders placed one on either side of the contracture. Large volumes of inflation are required both because the normal curvature of the saddle shape of the neck comprises a large surface area and because the wound edges will open up after excision of the contracture

resulting in a larger defect. If a superiorly based transposition flap is raised from the skin over one expander and an inferiorly based flap from the other these can be imbricated accross the front of the neck avoiding a vertical scar.

Even if only one side of the neck is spared considerable improvement can be made by producing an advancement flap with a unilateral tissue expander. This procedure may be repeated to completely resurface the scarred area of the neck.

Case Report 2

A 24 year old girl requested correction of scarring of the anterior and left side of her neck caused by a deep flame burn seven years previously. On examination there was marked scarring over this area which extended onto her chest, (Figure 2.6). Most of the skin on the left side of her neck had been spared. Although there was some limitation of extension the main problem was one of cosmesis.

A 300 cc. expander was placed subplatysmally under the expander of the left side of her neck and inflated to a volume of 460 mls. before an area of necrosis necessitated its removal,(Figure 2.7).

The expander was removed and an advancement flap raised which enabled excision of the scarred neck skin and the patient remains pleased with the final result,(Figure 2.8).

Figure 2.6 Case showing anterior neck scarring

Figure 2.7 Patient with inflated expander in neck showing a small area of necrosis before removal.

Figure 2.5 Result 6 months following reconstruction

Ear Reconstruction

Reconstruction of the ear amputated through trauma or absent in microtia has always been a great challenge to the plastic surgeon.

A considerable amount of thin pliable well vascularised skin of excellent local colour and texture match is required. In order to create the contour of a normal ear the skin must drape over an intricate skeleton and conform closely to it. Postoperatively there must be minimal contracture of the tissues as they heal lest the definition of the reconstruction is lost, (Brent 1980).

Tissue expansion has produced the opportunity of increasing the area of local skin around the missing ear. In addition the process of expansion thins the

subcutaneous tissues and in this site will produce a very thin pliable flap. Since Neuman's original case report there have been other reports using this method, (Sasaki 1986, O'Neal et al 1984), and in each case sufficient skin has been generated to cover the framework. The volume of saline injected which is necessary to produce sufficient skin to cover an ear was originally estimated at 60 to 100 ccs., (Sasaki 1986), however in practice greater volumes may be necessary, (Nordstrom 1988), presumably to take into account later shrinkage.

In none of the above reported cases is definition as good as in the normal ear. As the maximum follow up in these cases is only 6 months there must be a strong possibility that the reconstructions may later deteriorate due to gradual shrinkage of the expanded skin.

Long term clinical results need to be assessed before Tissue Expansion becomes the reconstruction of choice for the ear, especially in the light of recent advances in the field of fixation of facial prostheses, (Tjellstrom et al 1985).

Case Report 3

A 14 year old girl with a mild degree of hemifacial microsomia and microtia requested an ear reconstruction. Previously she had had a chin implant which was satisfactory and five operations to reconstruct a pinna which had achieved little except an area of scarring on

the left side of her face, (Figure 2.9). A further attempt at reconstruction was made at the insistence of both mother and patient and an expander was therefore placed subcutaneously at the proposed site of reconstruction and then inflated very gently over the course of four months to a volume of 220 ml., (Figure 2.10). At a further operation the expander was removed and replaced with an autogenous cartilage skeleton carved from her seventh rib, (Figure 2.11). Three months later a tragus was constructed and her contralateral ear set back. The appearance 6 months following reconstruction is shown in Figures 2.12 and in comparison with her normal ear, (Figure 2.13).

Figure 2.9 Left sided microtia prior to recent surgery

Figure 2.10 Full inflation of the expander.

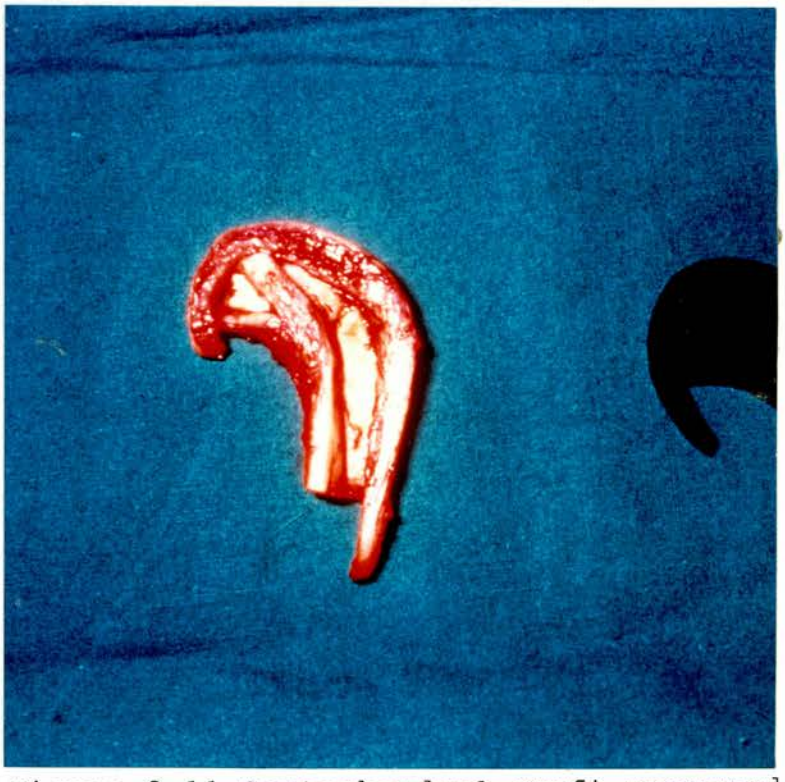


Figure 2.11 Costochondral graft prepared for insertion.

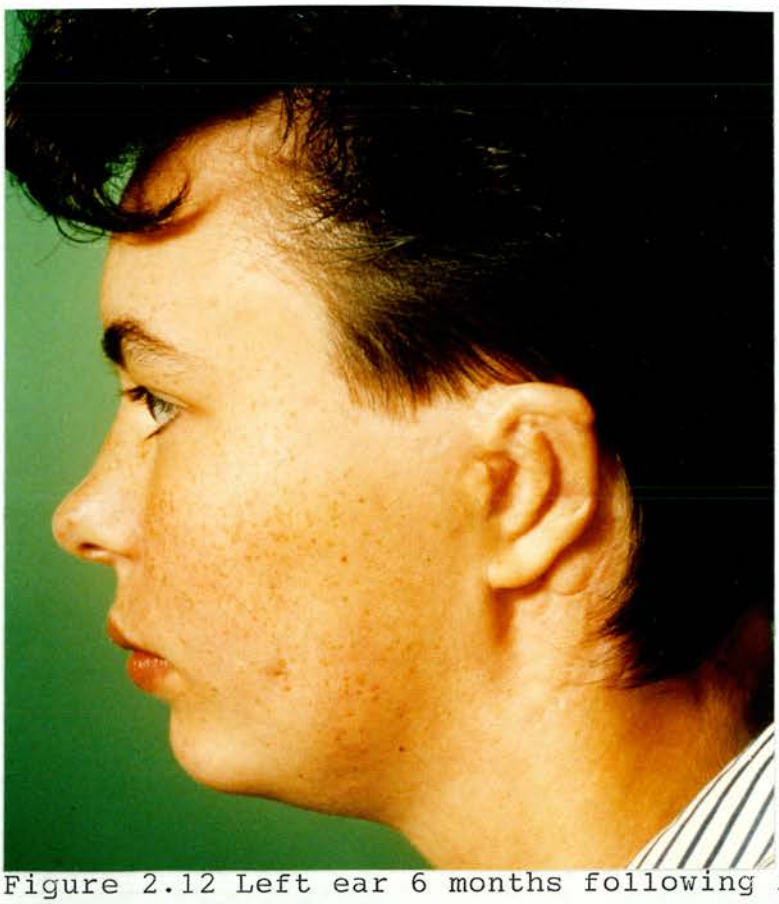


Figure 2.12 Left ear 6 months following reconstruction

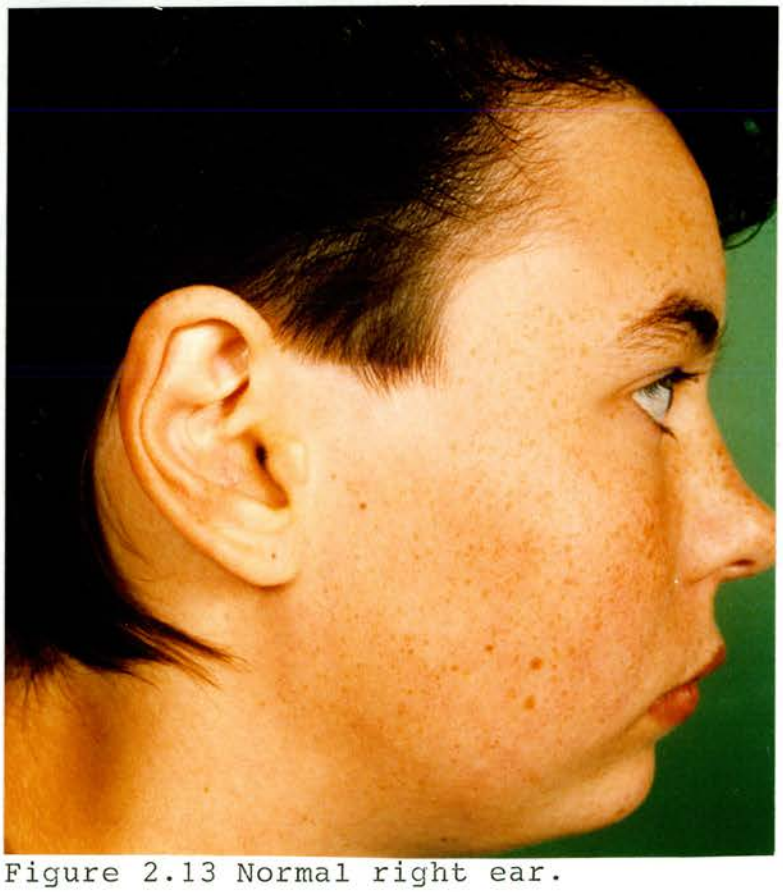


Figure 2.13 Normal right ear.

2.2.2. EXTREMITY RECONSTRUCTION

In the limbs expansion has most frequently been employed to enable the removal of damaged areas of skin. This may involve only the resurfacing of skin, for example a large tattoo, or in addition to the removal of an unsightly skin graft, the correction of a contour defect such as might follow trauma or melanoma excision. In a few cases it has been employed in acute trauma to achieve closure of wounds which would otherwise require alternative pedicled or free flap cover, (Manders 1988). There are however several reasons why tissue expansion is to be avoided in the closure of open wounds in the acute situation; most importantly the wound must remain open for a further four to ten weeks during the expansion phase and secondly there is a major risk of infection if the expander is placed adjacent to a contaminated wound. However once the wound is closed tissue expansion has proved itself to be useful in providing a more durable and aesthetically satisfactory cover. In some cases an additional benefit may be the replacement of an area of graft or scarring by an innervated flap - as might be required in an amputation stump, (Rees 1986).

The limbs are not the easiest areas for expansion and extra technical difficulties need to be considered. Patients with peripheral vascular disease and some metabolic conditions such as diabetes should be excluded because large areas of the limb will effectively be

degloved on insertion of the expander. Although a subfascial pocket is safer for the skin flap and easier for the surgeon to dissect, as cutaneous nerves for most of their length are superficial to the deep fascia, surgical elevation of a flap which includes the fascia will involve division of these nerves. In addition fascia resists expansion and consequently encourages atrophy in the underlying muscle compartments. For this reason most surgeons advocate subcutaneous placement of the expander in the limb and that if any large cutaneous nerves run across the planned flap that the pocket is dissected superficial to them, (Van Beek 1987).

The geometry of flap design is also influenced by the curvature of the limb. In the forearm and leg the radius of curvature is much smaller than in the head, neck or trunk and as a result a large expander placed in either of these sites will cover a large proportion of the limb circumference and thus there is a danger that the margin of a long flap raised round the limb will pass across its own base jeopardising its blood supply, (Godfrey 1984). For the same reason expansion will cause an increase in tension round the whole circumference of the limb. Intraluminal pressures of expanders in limbs are found to be higher than in other sites even at rest and only small increments in pressure may be possible at each inflation. Most problems occur in the distal limb and the complication rate is much less at sites more proximal in the arm and thigh, (Manders 1988 and Van Beek

1987). Here the skin is looser and the subcutaneous fat thicker allowing the expander to be well covered. Larges areas of skin may be resurfaced if there is unsufficient healthy skin to cover multiple expanders by the repeated expansion of a single site, (Sellers 1986).

Case Report 4

A forty year old lady requested improvement in the appearance of her anterior right shin. Fourteen years previously she had had a wide excision of a malignant melanoma from this site and the defect had been repaired by a split thickness skin graft. She thus not only complained of the scarring from the graft but of a contour defect also. In view of the long period following presentation during which she had remained disease free it was considered that correction of the defect would not prejudice her prognosis. A tissue expander was placed subcutaneously superior to the defect and inflated to 750ml, (Figure 2.15). Two months later the skin graft was excised and a transposition flap was raised from over the expander to resurface the defect, (Figure 2.16).

Figure 2.14 Expander inflated superior to defect.

Figure 2.15 Transposition flap transferred.

BREAST RECONSTRUCTION

Direct closure of wounds of the trunk is often possible despite excision of large areas of skin. In addition the trunk is most of the time covered by clothing and aesthetic reconstruction of this region is less frequently requested than elsewhere except for the one major exception of breast reconstruction following mastectomy.

Although it may be possible to insert a prosthesis beneath a mastectomy scar of a volume which equals that of the normal breast symmetry will not be achieved until the surface area of skin has been restored to match that of the normal side also. Conventional treatment involves the importation of skin by the use of flaps such as the Transverse Rectus Abdominis Muscle Flap or the Latissimus Dorsi Muscle Flap, (Hartrampf et al 1982, Scheflan and Dinner 1983 and Bostwick et al 1978).

Radovan first presented his results of a series of breast reconstructions in 68 patients in whom he had expanded the remaining chest wall skin to a larger than the desired size by a temporary expander which he had later replaced with a smaller implant to match the contralateral normal side. Since then more has been written on the use of Tissue Expansion for breast reconstruction than for any other clinical problem.

For a woman who has already suffered the physical

and psychological trauma of a mastectomy Tissue Expansion has a major advantage over other methods of reconstruction in that the surgery is of limited magnitude and virtually no extra scarring is involved. Considerable experience in this method has thus been accumulated and some problems have become apparent. The more tissue that has been left at the mastectomy the safer is expansion and the better the final result. Scarring is the main factor which impedes skin expansion and worst of all is scarring from radiotherapy; expansion is slow and extrusion common, (Dickson et al 1987).

As with the insertion of breast prostheses for breast augmentation capsule formation can be a problem. Capsular contracture following breast augmentation will spoil the result by making the prostheses feel hard and causing their outline to be visible; contracture following an expanded breast reconstruction will cause the whole breast to lose both projection and ptosis and to feel unnaturally hard. As the capsule contracts around the permanent prosthesis the skin envelope is no longer stretched out to length by its contents and it will shrink with the result that the increase in skin area due to expansion is partly lost.

In his initial article on the subject Radovan advises that both expander and permanent prosthesis should be placed in a superficial subcutaneous position in order to minimise capsular contracture, (Radovan 1982). Other surgeons disagree and advocate a submuscular

placement beneath pectoralis major if this is present following mastectomy, (Argenta 1984 and Gibney 1984). This would concur with the reduced incidence of contracture following submuscular breast augmentation, (Mahler and Hauben 1982) and in addition appear to be a more favourable location to prevent expander extrusion.

Another factor which is thought to influence the final area of the skin envelope is the volume to which the expander is inflated. It is now accepted that the expander should be overinflated for a period before exchanging it for the permanent prosthesis. After a large series of reconstructions it has been found that overexpansion by a factor of 50% for 2 months prior to exchange with a normal prosthesis to be satisfactory, (Gibney 1986).

In addition to techniques designed to increase the surface area of the skin envelope an alternative way by which the appearance of ptosis is produced is to create a definite infra-mammary fold which is often absent after tissue expansion. This can be achieved by hitching the deep surface of the skin up to the ribs at the proposed site of the crease. The undersurface of the skin crease may be approached either through the mastectomy wound when the expander is replaced by the permanent prosthesis or or by making a fresh incision along the crease and advancing a flap of de-epithelialised abdominal skin which is sutured up to the ribs, (Ryan 1982). Either technique will create a definite fold but adds significantly to the extent of the surgery.

As with other methods of breast reconstruction in order to improve symmetry of size and shape it is often necessary to perform a breast reduction or mastopexy of the normal breast.

In summary in most cases a breast mound can be formed by this method though there is a difference of opinion amongst surgeons as to whether expansion produces as good a result as produced by other methods. Breast reconstruction is normally an emotive subject for the patient concerned and as it is not an essential part of treatment it is perhaps wise, after a full discussion of all possible options, to allow the patient to take a major part in deciding which method is most suitable for her needs.

Case Report 5

A 48 year old lady requested reconstruction of her right breast, Figure 2.16. Nine years previously she had had a mastectomy for a spheroidal cell carcinoma along with an axillary dissection from which most of her nodes were positive. Four years following the mastectomy she had radiotherapy for axillary recurrence but has remained disease free till present. The mastectomy scar was excised and a subpectoral pocket dissected and a tissue expander inserted, Figure 2.17. This was inflated to 500 cc. over a period of three months, Figure 2.18, before being replaced by a permanent 320 cc. implant. At this operation an inframmary crease was created by anchoring

the flap to the 7th rib, Figure 2.19. Finally after an interval of three months a mastopexy was performed on the contralateral breast and the opportunity was taken to redissecect the pocket in the right breast a little further medially to improve symmetry, Figure 2.20.

Figure 2.16 Right Mastectomy prior to reconstruction

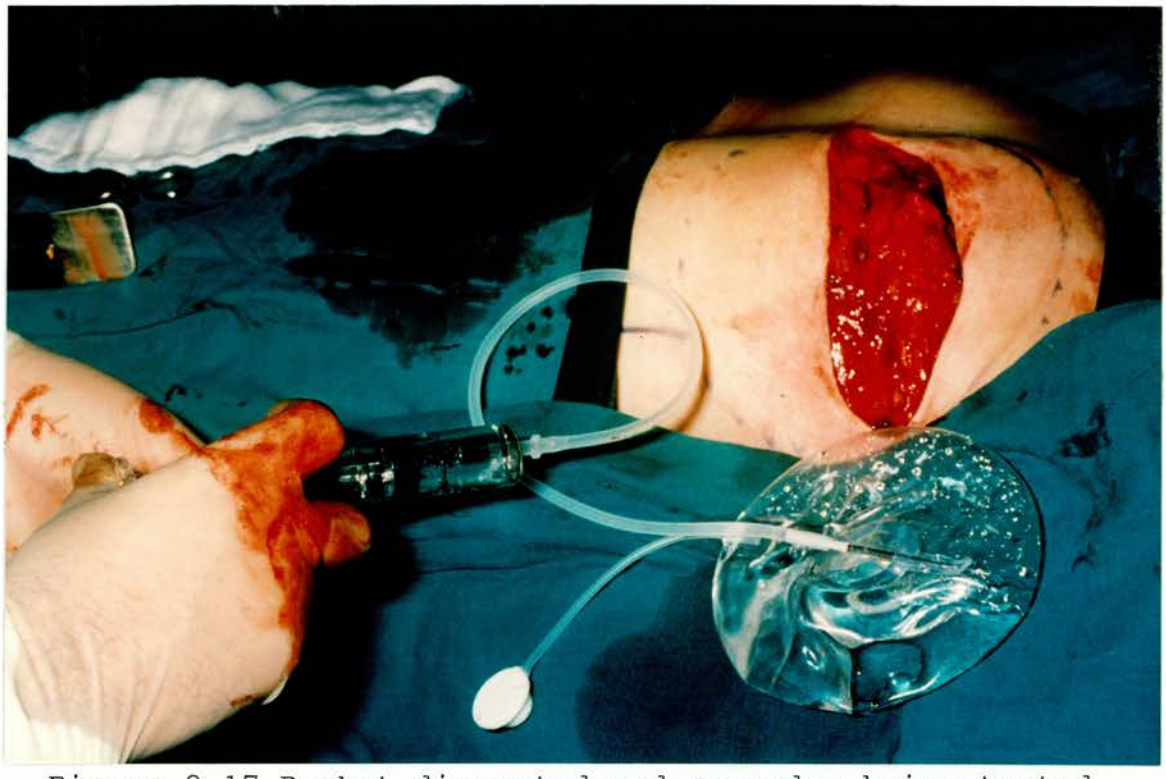


Figure 2.17 Pocket dissected and expander being tested

Figure 2.18 Expander right breast overinflated

Figure 2.19 Permanent right implant and left mastopexy

Figure 2.20 Good symmetry in brassiere

2.3 COMPLICATIONS OF TISSUE EXPANSION

Even in the hands of enthusiasts a significant complication rate is reported with this technique which is usually in the order of 40 %, (Manders 1986, Austad 1988). However despite this high rate expansion is often successfully completed and a satisfactory reconstruction achieved in over 80% of cases, (Antonyshyn 1988). Complications which require the procedure to be aborted are referred to as major and those despite which expansion can continue are referred to as minor, (Austad 1988).

In addition to problems specific to Tissue Expansion the patient is also at risk of normal general surgical complications. At insertion of the expander a haematoma may develop around the expander. Most surgeons will routinely use a suction drain to remove blood produced by the initial dissection. As with the insertion of any prosthetic device infection is disastrous and will almost always lead to loss of the device. Thus prophyllactic antibiotics are also frequently given.

2.3.1.Implant Failure

There is a wide variety of implants available "off the shelf" produced by a number of companies. The frequency of implant failure will thus depend on the quality of that particular type of device because the design specifications and in particular the strength of

the silicone will vary from manufacturer to another. Failure of an implant will normally result in rapid deflation of the expander and this is usually due to rupture of the junction between the connector tubing and the expander. Rupture of the expander wall itself is uncommon since the envelope of most devices is capable of being expanded many times the recommended volume, (Hallock 1987). Salvage of implant failure is only possible by surgical re-exploration and replacement with a new device.

On occasion the surgeon may suspect a slow leak in the system if the expander feels extremely slack before each inflation and very large injections are required to make it turgid again. In the absence of accidental puncture of the balloon this is almost certain to be due to leakage of saline back out through the puncture holes caused by the needle entering the valve. Most expander valves are designed to be self sealing due to layer of silicone gel in the valve which blocks off the needle hole. Despite this leakage is possible from the system if the pressure within the expander lumen is significantly increased such as would occur if the patient lay on the expander,(Nordstorm 1988). On removal of an expander it is not uncommon to find that its weight full of saline is considerably less than that expected from the total weight of saline injected and thus valve leakage is probably much more common than appreciated.

Difficulty in inflation is much more likely to be

due to surgical error than implant failure. It is important that the needle is placed into the depth of the valve against the backplate for fluid to flow into the expander. Too superficial a puncture will place the needle tip in the silicone gel of the valve and prevent inflation, (Yang 1988). Inflation will also be difficult if there is a kink in the connector tubing - as may occur if this is looped due to it being too long for the distance between valve and expander.

2.3.2.Problems during expansion

Before starting reconstruction the surgeon is advised not to promise the patient a completion date for the procedure, (Argenta 1984), because different skin will expand at different speeds. In some patients the gain in skin area may take much longer to achieve than expected. Irradiated skin is particularly resistant to inflation, (Dickson 1987), and expansion of this is virtually contraindicated. Many problems are related to the rate of expansion - this is a product of the time allowed for wound healing before expansion commences, the frequency of inflation and the volume of saline injected on each occasion. No studies have been published which define the optimum rate of expansion and at present it depends on the preference of the individual surgeon.

In patients in whom expansion is slow pain is often the cause which limits the volumes injected on each occasion. This may be due to stretching of the capsule or of overlying tissues such as muscle.

It is normal for pain to settle within a few hours of inflation as the skin relaxes but if it is intolerable fluid will need to be aspirated from the expander which will lengthen the time required to achieve the necessary skin area.

Apart from infection around the expander the most serious problem that occurs during this phase is exposure of the implant. This may be caused by several factors all of which produce ischaemia of the overlying skin. Very aggressive tissue expansion will substantially thin the subcutaneous tissues and make the skin more susceptible to pressure necrosis from the expander beneath. This problem may be exacerbated in a localised area of the flap if there is a fold or "knuckle" in the wall of the expander. Knuckles usually appear early on in expansion and are caused by failure to "iron out" all the creases in the expander on its insertion, often because the expander is too large for the subcutaneous pocket dissected. Attempts have been made at unfolding the envelope by aspirating saline from the system and then manipulation, (Antonyshyn 1988), but this seldom works. A more effective solution is to firmly tape the skin down over the knuckle with inelastic tape . This has the effect of inverting the point in the expander wall allowing inflation to continue, (Saxby 1988).

Other causes of exposure are erosion of the valve through the skin and wound dehiscance by inflation too early and too aggressively. If exposure of the device

does occur it does not necessarily mean that the procedure must be abandoned. An area of exposure will increase in size if inflation continues, (Austad 1985), but if it occurs towards the end of expansion it is possible that the procedure may be completed with no ill effect, (Argenta 1987). If it occurs early on the expander should be removed, the cavity irrigated with antibiotic solution, the wound causing the exposure excised and closed in layers and the expander reinserted.

In some cases it is the patients themselves who cause exposure due to self mutilation. In a study on patients undergoing mastectomy reconstruction two women had intensely disturbing psychological reactions to their tissue expansion (Goin and Goin 1988) and careful preoperative councilling is essential.

Several other problems that occur in this phase are due to the local effect of pressure on tissues. Fat atrophy is often seen - this is not usually a major problem but as it is permanent it may detract from the final result. Atrophy is also seen in bone and muscle but there is not usually a long term problem. Neurapraxia of cutaneous nerves that run in the skin flap sometimes occurs, usually in limbs, and this results in an area of anaesthesia distal to the expander. This invariably resolves after removal of the expander.

The force involved may be enough to rupture the transverse collagen linkage in some patients' skin and the result of this is the appearance of striae which may

permanently reduce the aesthetic quality of the final reconstruction.

Finally inappropriate areas of skin may be expanded if the expander sinks inferiorly due to gravity or moves to an area of less resistance. The latter is seen when an expander herniates from beneath irradiated skin to surrounding non-irradiated tissue. In either case there is little that can be done except adjust the skin at the time of reconstruction.

2.3.3.Late Complications

Following definitive reconstruction as with any flap transfer there is an incidence of flap necrosis. This may be due to excessive tension or poor vascularity within the flap either of which is likely to be due to poor flap design.

In procedures designed to resurface adjacent defects complications are normally minor as, once wound healing has taken place late skin shrinkage will have little adverse effect. Scars may widen with time but this is no more of a problem as in nonexpanded skin. Dog ears of redundant skin are not normally excised primarily as considerable shrinkage can be expected and if they do not disappear completely they can be removed at a later stage by a much smaller excision.

On the other hand expansion designed to produce three dimensional reconstructions may suffer considerably from late shrinkage of skin. If skin has been draped over

a very intricate framework such as in ear reconstruction this problem may result in a very disappointing loss of detail months later. The effect of shrinkage on the skin envelope in breast reconstruction has already been discussed. This problem is an inherent part of tissue expansion and apart from prolonged overexpansion no satisfactory solution has been achieved. It is hoped that work directed towards achieving permanent skin growth rather than skin stretching will reduce this problem.

2.4 SUMMARY

Tissue Expansion is a technique for generating skin flaps by gradual distension of skin. It differs from other similar methods which involve distension, e.g. serial excision, in that the skin required is distended prior to reconstruction.

The gradual expansion is achieved by the insertion of an inflatable device which can be serially inflated at intervals of a few days by percutaneous injection of saline. Considerable clinical experience has been gained in the last few years in the treatment of many different conditions in almost all sites on the body. One of the main advantages of this technique is that as the skin used for reconstruction is normally adjacent to the defect it is likely to be of very good colour and texture match. In some sites, e.g. the scalp, Tissue expansion has become the reconstruction of choice but in others there are inherent technical problems which may render it impractical.

Generally the complication rate is high and this probably reflects the first part of a learning curve in which the technique has been tried for many varied problems before enough clinical experience has been acquired to allow Tissue Expansion to find its place in the plastic surgeon's armamentarium.

CHAPTER 3 PATHOPHYSIOLOGY OF TISSUE EXPANSION

Tissue Expansion involves three pathological processes and these will be discussed in detail.

3.1. CREATION OF A SUBCUTANEOUS POCKET AND INSERTION OF EXPANDER.

In order to insert a tissue expander a skin incision is normally made along the margin of the proposed skin flap and then the flap is undermined in order to create a subcutaneous pocket large enough to accommodate the empty expander. It has long been known that to incise the margin of a skin flap and or undermine the flap will have an effect on its ability to survive after being raised. In the middle of the fifteenth centuary surgeons from the Vianeo family refined the Branca method of nasal reconstruction, which uses a flap of skin from the arm, by undermining the flap and separating it from its bed by insertion of a piece of medicated linen one month prior to transfer,(Gnudi 1951 and Webster 1959). Such a procedure, performed a week or two prior to raising a flap with the intention of increasing its survival after transfer, is popularly known as a "Delay", first being described as a "Delayed Transfer", (Blair 1921). Various forms of delay of flaps were investigated in a series of

flaps on pigs' flanks, (Milton 1967). It was discovered that by incising three margins of a rectangular flap three weeks prior to its elevation that the surviving length could be increased by as much as 13% when compared to a similar flap which was elevated acutely. A further improvement in survival was achieved by incising the flap along two adjacent borders and undermining the whole flap When performed two weeks prior to elevation this enabled a surviving length 53% greater than in the comparable acutely raised flap. The increased survival achieved by this method of delay was explained by the fact that a much larger number of supplying blood vessels would be divided by interrupting the deep perforating vessels when undermining the flap than by incising along its margins. Milton concludes that the most effective form of delay is one which divides the greatest number of vessels supplying the flap (excluding those which will become part of the pedicle) without causing necrosis.

Considerable research has been performed in order to identify the mechanism by which a surgical delay permits greater flaps survival than would otherwise be possible. An understanding of the mechanism is also likely to explain why necrosis may occur in a flap. Theoretically it would be possible to mimic surgical delay in a flap by pharmacological or electrical manipulation either to increase flap safety or even reverse undesirable changes in a flap which appears to be failing.

Although surgical delay is less commonly used today

it was an integral part of tube pedicle transfer and thus much work was performed on the subject with reference to changes that occurred in this type of flap. An early explanation for the beneficial effect of delay was given by Gillies, 1921 and 1939, who suggested that "the first parallel incisions cut off horizontally inclined blood vessels so the flap depends only on the longitudinal anastomoses". Following this the horizontally arranged anastomoses would atrophy and the longitudinal ones would hypertrophy resulting in a reorientation of the blood flow in the skin of the flap. These changes have been found and described in detail, (German et al 1933, Braithwaite 1950,1951). If the vessels are already arranged in a longitudinal fashion in the flap no such changes are seen, (Bellman and Velander 1959), and this would concur with the suggestion that for delay to be successful major vessels supplying the flap outwith the pedicle must be divided, (Muir et al 1968).

Although angiogenesis is postulated as the means by which these vascular adjustments are achieved as the maximum changes occur in two to five days this is unlikely and it would appear that it is the existing vascular network which alters, (McFarlane 1965). When the vessels crossing the margins of the flap are surgically divided during a surgical delay procedure they also suffer sympathetic denervation because the sympathetic nerves travel along the vessel walls. It would follow that this denervation would affect most of the flap and

reduce the tone of the vascular tree thus decreasing its peripheral resistance, (Hynes 1950). As a result blood from a higher pressure system from without the flap entering through the pedicle would flow further into the flap than it would have done if the blood vessels maintained their sympathetic tone. Sympathetic denervation was demonstrated by mapping out affected areas using a sweat test.

Although vascular reorientation is an attractive theory it would not fully explain the effect of the delay phenomenon because it is seen very early on and reaches a maximum well before the delay confers the possibility of raising a larger viable flap. Furthermore the vascular changes appear to regress and after two weeks the vascular pattern may return to normal in some instances despite the effect of the delay persisting, (McFarlane 1965).

It becomes difficult to interpret evidence for alternative theories because different workers have drawn very different conclusions. In McFarlane's experiments he found that there was no increase in blood supply to the distal end of a flap raised after delay compared with a comparable flap raised acutely. Using uptake of radioactive rubidium, which behaves like potassium, as a marker of cell metabolism and thus tissue hypoxia he found that the distal ends of both delayed and acutely raised flaps were equally hypoxic. Since that part of the delayed flap survived when the comparable part of the

acute flap necrosed he concluded that the effect of delay was to condition the tissue to survive hypoxia due to ischaemia.

Absolute blood flow entering a flap is not necessarily the factor which determines flap survival. Obviously if the vascular network is not orientated in a manner which allows blood flow into the distal end of the flap there will be incomplete survival. Hence an increase in blood flow does not necessarily produce increased flap survival, (Neligan 1985). Even if blood does reach all parts of the flap it needs to flow through the capillary bed to prevent ischaemia. Reinisch, 1974, measured blood flow at centimeter intervals along recently raised flaps and found significant blood flow right to the end of a flap which was only partially viable as estimated by flouroscein staining. He then investigated nutrient blood flow only by measuring radioactivity levels in a similar flap after injecting 15 micron diameter microspheres which would only become trapped in capillary beds. He found this nutrient blood flow to be confined only to the viable proximal part of the flap and concluded that the blood flow to the distal part of the flap was directed through open arterio-venous anastomoses, i.e. shunted, and had no beneficial effect on the skin. He suggested that opening of these A-V shunts would also occur as a result of a flap delay but that within two weeks these vascular communications would regain tone and close down preventing this phenomenon from damaging the flap when it

was raised. These experiments have been repeated but the results have not been confirmed by other workers, (Kerrigan 1983). In the most recent reports on the subject the total skin blood flow and nutrient blood flow has been found to increase in flaps as a result of delay, (Pang 1986), and there was no increase found in the proportion of non-nutrient blood flow which passed through A-V shunts, (Kerrigan 1983 and Guba 1980). A further hypothesis is that the vascular changes are mediated by the consumption of vasoconstrictive neurohumeral factors produced by the trauma of delay and vasodilator substances produced by the subsequent inflammatory response, (Pang 1986).

Thus the exact nature of surgical delay remains a mystery and in clinical practice its use is empirical. There has also been debate about the optimum period of delay necessary to confer the greatest benefit to a flap. It appears to depend on the type of surgical procedure being performed. For delay by undermining a bipedicled flap two weeks is sufficient but for incision around three margins of the flap with no undermining three weeks is necessary, (Milton 1967). It may also differ according to species, (Myers 1971). As these studies are performed by raising flaps long enough to ensure some distal necrosis enabling the measurement of surviving lengths no such comparisons can ethically be made in humans.

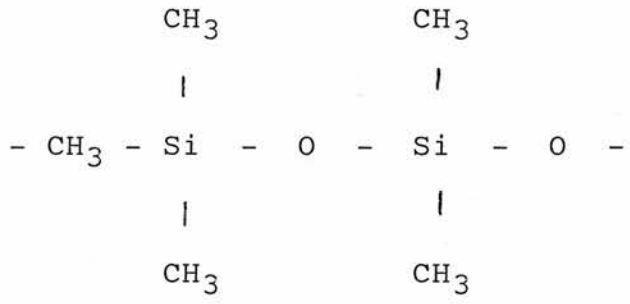
When the flap is laid back in its bed after the delay the process of wound healing occurs with the result

that new vessels grow into the flap across the wounds so that after a period of two to three weeks the delay starts to lose some of its effect, (Myers and Cherry 1967). This situation is not completely analogous to the insertion of a tissue expander because the presence of the silastic envelope separates the undermined flap from its bed preventing ingrowth of vessels into the flap in all but around the edges of the expander.

In summary as result of these factors the insertion of a tissue expander acts as a potent delay increasing the viable length of flap that may be raised, regardless of any effect produced by hydraulic distension. The delay should be effective as long as the expander is present to separate the flap from its bed, i.e. to the completion of expansion when the flap is utilised for reconstruction. This has been confirmed by Cherry et al,(1982), who have compared survival lengths in flaps which have had expanders placed subcutaneously and left empty for five weeks before being raised and in flaps raised acutely. An increase of 47% in surviving lengths of the flaps "delayed by expander" was found.

3.2. CAPSULE FORMATION AROUND SILICONE ELASTOMER IMPLANTS.

The term silicone was first used by F.S.Kipping to describe a group of compounds based on a polymer of the dimethylsiloxane radical :



By varying the proportions of the components of the molecule the silicones can be made to range from volatile liquids to dense solids. Although their original use medically was external as a component of appliances and dressings it was postulated that they might have a major application as subcutaneous implants, (Barret Brown 1953). A further seven years elapsed before a detailed report was made of the results of their subcutaneous implantation in clinical cases, (Barrat Brown 1960). Very rapidly silicone elastomers have found a place as the material of choice for a wide ranges of prosthetic implants in particular for breast augmentation, facial contouring and joint replacement.

The acceptability of this material is mainly due to the low response produced by the host tissues. Silicones

are not however inert and a very characteristic reaction occurs within the host tissues at the interface with the implant. A fibrous layer is formed which surrounds the foreign body and is known as a capsule. Complications which follow implantation of silicones are often related to the capsule which is formed in every case but only becomes a problem in a percentage.

The envelope of a tissue expander is made of a similar elastomer to that which surrounds gel filled mammary prostheses which have been in use for over 25 years. Following breast augmentation with such implants capsular contracture is the most frequent complication, occurring in up to 50% of cases, (Vinnik 1976), and thus the structure of these capsules have been studied in detail, (Domanskis 1976 and Ginsbach 1979).

The natural history of the capsule has been studied in the guinea pig following insertion of small blocks of Silastic*, (Thomson 1973). Eight days following implantation a cellular pseudosheath develops which is largely comprised of fibroblasts which show only a little polarity. Within a month there is gross polarity in the fibroblasts and the capsule becomes a little thinner. By two months the cellular component is reduced and there is a much increased amount of collagen, in addition capillaries and nerve fibres are seen within the capsule. As further time elapses the capsule becomes thinner and becomes infiltrated by fat. The capsule is not a permanent entity and will disappear a month or two after

the removal of the implant. Studies in the human reveal that the structure of the capsule is not uniform throughout its depth. The luminal surface of the capsule is often seen to have an epithelial-like layer. Although this is not always seen it is only loosely attached to the deeper structures and is often lost during processing thus its absence is probably artefactual. The middle layer of the capsule is found to be a very homogeneous layer consisting of collagen fibres orientated parallel to each other and to the surface of the implant. Amongst the collagen are interspersed cells which, under the electron microscope, have the characteristic features of myofibroblasts. At a more superficial level the collagen fibres lose their orientation and become less dense and interwoven giving the tissue an areolar texture, (Ginsbach 1979).

Much effort has been expended trying to determine why some capsules cause problems by contracting around the implant. If this occurs following breast implantation it imparts a hard lumpy texture to the breast and, if severe, a visible alteration in contour. If it occurs during tissue expansion it may produce great resistance to the hydraulic distension of the implant. There have been various hypotheses to explain the cause of this problem involving glove powder, haematoma and low grade infection but the explanation that is most popular is that there is an escape of silicones from the device into the surrounding tissues, (Gayou 1979). In many cases

minute droplets have been found in the capsules surrounded by a cellular reaction and having the characteristics of silicones. The envelope of these devices is not impermeable, (Rees 1968), and many substances can diffuse through including silicone gels themselves. It is not uncommon to find that breast prosthese have an oily covering when first removed from the packet and this is due to the "bleeding" of silicones through the wall. As the droplets are also seen in capsules around saline filled devices they may originate from the wall of the implant also, (Domanskis 1976).

As all capsules probably contract to some degree it is difficult to identify the causative factors in those which are a problem. It appears that contraction is not related to amount of droplet formation in the wall or foci of inflammation which may occur. The only striking change that is seen in capsules which are grossly contracted compared to those which are not is an increase in the cellular component. Although there are only a few myofibroblasts seen and not enough to cause the response experiments have shown that they may be able to "set the tension" in the surrounding collagen fibres, (Baker 1981).

Some evidence is accumulating to suggest that the effect of hydraulic distension will modify the structure of a developing capsule. Not only are capsules around expanders thinner than their counterparts around static prostheses but there also appears to be a change in their

ultra-structure; the expanded capsule has a reduced collagen component and is more cellular. The increased cellularity is mainly due to an increased number of fibroblasts but there is also an increase in the number of blood vessels, (Laitung 1987). It is claimed that there is an increase in the proportion of type III collagen which is characteristic of the early stages of scar formation before a permanent stable structure is produced, (Shuttleworth et al 1978) .

Although a higher density of blood vessels is seen in the capsule the blood supply to the capsule is unlikely to be increased. Electron microscopy reveals thickening of the endothelial basal lamina suggestive of degeneration, (Pasyk 1982) and thus ischaemia may be responsible for changes seen in a capsule in the final stages of its maturation, (Arons 1961).

3.3. EFFECT OF HYDRDAULIC DISTENSION

3.3.1. Changes in tissue structure.

A skin flap is comprised of three basic layers: epidermis, dermis and subcutaneous fat but the behaviour of skin when subjected to mechanical forces is mostly related to the structure of the dermis. This layer can be divided into two according to the arrangement of the collagen fibres. The papillary dermis is a thin layer which lies immediately beneath the epidermis and is comprised of fine collagen fibres which form a dense woven network and beneath this is the thicker reticular dermis where thicker but more sparse bundles of collagen fibres are arranged in an interlacing fashion parallel to the skin.

The response of skin to a distending force can be divided into viscoelastic and biological changes according to the duration and magnitude of the force, (Gibson 1977). Elongation of skin, known as creep, can be produced in minutes by the application of distracting forces in the region of 1,000 lbs. per square inch. This increase in length occurs due to the mechanical deformation of collagen fibres within the dermis, (Gibson, Kenedi et al 1965). Normally the collagen fibres within the dermis are arranged in a loose convoluted lattice but when stretched the convolutions straighten

out and as the force is increased the fibres become aligned. It has been found clinically that the amount of skin creep can be increased if the force is not continuously sustained but used intermittently which was described by Gibson as load cycling. The amount of skin area gained is limited because when the collagen fibres are aligned no further extension is possible, (Hirshowitz 1986). Forces of this magnitude cause blanching of the skin due to the blood vessels within the dermis being constricted when caught up in the reorientation of the collagen fibres which will eventually lead to tissue necrosis. Thus load cycling can only be used for short periods of time though it remains a useful tool in the closure of wounds which are tight. Conventionally this was achieved by placing sharp hook retractors into the under surface of the dermis on either side of the wound and pulling them very forcibly together. This has been refined in the technique of intra-operative expansion which involves the placement under anaesthesia of one or more expanders adjacent to a wound which is too large to close directly. The expanders are inflated to high pressures for several periods of approximately five minutes leaving time in between expansions for the skin to perfuse. If planned correctly the distension of skin produced may permit closure of large defects without tension, (Sasaki 1988 and Pietila 1988).

Some reorientation of collagen fibres may occur in conventional tissue expansion. This could be the cause of

the reduction in tensile strength found by Schneider et al, (1988). However the viscoelastic properties of skin do not alone explain the changes that occur. The response of skin and subcutaneous tissues to distending forces of lower magnitude but much longer duration is by biological mechanisms which are, as yet, poorly understood.

The structure of skin following expansion has been examined in both patients and animal models, (Pasyk and Austad 1982,1986 & 1988, Johnson et al 1988). The cellular response within skin that is being expanded is limited, there being little sign of inflammation and little cellular infiltrate outside the capsule which has already been discussed. However certain changes consistently occur. After a period of expansion the thickness of the epidermis is seen to increase compared to non-expanded control skin. The mitotic activity of this layer has been assessed and found to be significantly increased during expansion, (Austad 1986). It was postulated that this was evidence that expansion caused an actual generation of new tissue rather than a thinning out of existing tissue as it is distended. In a study specifically concerning skin stretching and epidermopoiesis, (Francis and Marks 1977), a similar increase in epidermal thickness was initially found after stretching with a concomitant increase in epidermal mitosis. It was realised that some degree of inflammation was occurring in the skin and when the experimental model was modified to exclude this no increase in epidermal

thickness was found although the mitotic rate was still slightly raised. It was concluded that as a result of the loss of contact inhibition between cells proliferation only occurred at such a rate that would relieve tension.

Epidermal mitosis is however labile and easily provoked by stimuli such as minor mechanical trauma which is most likely to occur over the dome of an expander, particularly in an animal, (Bertsch 1976), and thus these causes are more likely to be responsible for the changes in epidermal thickness than the expansion itself. Furthermore the keratinocytes have a considerable capacity for regeneration if lost. As long as some epithelial cells remain this layer regenerates quickly as is seen following a superficial burn. Thus if the skin area is increased by mechanical distension it may be assumed that these cells have the capacity to multiply in order to cover the increased area. Thus it is unlikely that the epidermis will be a critical factor preventing a gain in skin area during tissue expansion.

A more important observation of change in epidermal structure was made by Van Rappard, (1988), who noted that the height and frequency of the dermal papillae was reduced giving the appearance of a flattened interface with the dermis. Thus regardless of keratinocyte mitosis the existing structure of the epidermis appears to be elongated by expansion rather than there being the formation of new papillae.

In comparison thickness of both dermis and

subcutaneous tissues are both seen to be reduced during expansion. Pasyk,(1982), found large bundles of compacted collagen within the dermis using the electron microscope which were thought to be present as a result of hypertrophy due to the sustained increase in skin tension although the total dermal thickness was reduced. No such change in collagen architecture was found in a similar histologic study of expanded pig skin, (Johnson et al 1988).The reduction in subcutaneous tissue thickness is brought about by atrophy of fat and muscle and reduction of the intercellular spaces. It is interesting to note that when examining skin from clinical cases which has been expanded and then used for reconstruction two years previously Pasyk found measurements of thickness of the epidermis, dermis and subcutaneous tissues not to be significantly different to measurements made before expansion. Thus even if there is largely a sharing out of dermis and subcutaneous tissues during expansion if the increase in area of an expanded flap is maintained and the flap regains the normal thickness of its component layers at some time there must be generation of new tissue. Recently it has been claimed that a net gain in collagen and hence a theoretical gain in dermis has been shown, (Johnson 1988), and certainly there is a net gain in the excess skin produced during obesity where the skin thickness is maintained, (Black et al 1971).

The origin of the increased area of skin is further obscured by the possibility of migration of local skin

into the field of expansion, known as recruitment. This can be studied if the margins of a flap are tattooed prior to expansion so that the migration of any skin from outwith this area over the expander dome might be identified. It has been shown in pig flaps that there is only a small degree of local skin recruitment which contributes to the skin cover of a full expander. However the skin around the periphery of an expander expands to a much less degree than that over the centre of the expander which is responsible for the vast majority of the increase in skin area, (Vander Kolk et al 1987). There is no doubt clinically that recruitment is a factor in generating area and it appears likely that the looser the surrounding skin the more likely this is to happen thus it is seen in the neck but less so on the trunk.

3.3.2. Survival of Expanded Skin.

For a flap to survive it is necessary for blood to perfuse the tip of the flap despite the division of many blood vessels during flap elevation. The route taken by blood when entering the flap will depend on the type of flap raised, (i.e. whether axial or random pattern, myo- or fascio-cutaneous) however once in the subcutaneous tissues of the flap it is conveyed through an interconnecting network of vessels. These are loosely

arranged in vascular plexuses although there is much intercommunication between them. Within the papillary dermis lies the subpapillary vascular plexus which is fed from and drains to the deep dermal plexus which itself lies at the interface between fat and reticular dermis. A third vascular plexus lying within subcutaneous fat at a level corresponding to the paniculus carnosus has also been found, (Pearl 1983), which, it is suggested is of importance in flap survival although this particular plexus would obviously be at risk of damage on insertion of the expander. Following tissue expansion it is the ability of these plexuses to convey blood further than in the nonexpanded state that enables an increase in flap survival.

Investigation of flap survival requires measurement to be made of the length at which flap failure and necrosis occur and would be unethical on the human subject. Relatively little experimental work has been performed in animals on skin expansion but that which has will be discussed.

Basic studies on the survival of expanded skin have been performed, (Cherry et al 1983 and Sasaki and Pang 1984). In both series of experiments dorsally based flaps on the flanks of pigs were used. Comparisons were made between the surviving lengths of flaps which were raised acutely, flaps which had previously been subjected to a conventional bipedicled delay, flaps which had been previously subjected to a "delay" by insertion of a

tissue expander which remained empty and flaps which had been subjected to tissue expansion. In these random pattern flaps it was not surprising to find that all flaps which had undergone prior surgical manipulation had longer surviving lengths than those flaps raised acutely. However in both series the flaps which had been expanded had significantly greater surviving lengths than even delayed flaps, there being no difference in flap survival between the two types of delay, conventional or by the insertion of an empty expander, (in Cherry's work the expanded flaps were 117% greater than an acutely raised flap compared with an increase of only 73% in those flaps which had under gone a conventional delay). In each series a period of 5 weeks was taken for expansion and total volumes of 100 and 250 ml. were used. This must be considered very gentle tissue expansion for an area such as the flank of the pig. As will be seen many times this volume could have been inflated and if this was the case it is likely that the survival rates in expanded skin could be shown to be greater still.

Following his survival studies Cherry performed microangiograms on the skin flaps and concluded that there was an increase in number and calibre of small blood vessels. This has been used as experimental proof of the hypothesis put forward by Radovan, who observing erythema in skin undergoing tissue expansion, suggested that angiogenesis was stimulated by this technique which would cause an increase in skin perfusion.

Several factors have been postulated as potential stimuli for angiogensis. At least two peptide hormones have been identified and one, Endothelial Cell Growth Factor, which, injected into skin preoperatively, will increase survival in skin flaps, (Hom et al 1988). Physical distention has also been implicated and traction on chick allantoic membrane has been shown to induce its microcirculation, (Jakob 1978).

In the early stages of angiogenesis solid rods of endothelial cells are seen to develop which later canalise to form a network of vessels, (Simpson et al 1983). These features have not been identified but as new vessels develop quickly in 48 to 72 hours and will arise amidst existing vessels it may be very difficult to identify them.

Some observations on blood flow in expanded skin flaps have been made, (Sasaki 1984). In this study identical experimental and control flaps were raised as before but instead of assessing them a week later to ascertain viability the pigs were given an injection of radioactive microspheres 16 hours after flap elevation. The flaps were then excised and divided transversely into sections 1 cm. wide. Radioactivity was then assessed in each section with a gamma counter and the following conclusions made. Perfusion appeared to be increased in expanded flaps and in flaps delayed either conventionally or by the insertion of an expander which remained empty when compared with flaps acutely raised. In addition the

presence of radioactivity was found further distally in these groups than in the acute flaps. No difference could be found however between the expanded and merely delayed flaps.

During the course of this project further studies were published on the effect of tissue expansion on blood flow. Leighton et al, (1988), reported a study in which blood flow was found to be increased in porcine skin flaps which had undergone expansion over a similar period of time as used in this study. As the experimental model was very similar to that used in this study it will be discussed in greater detail later.

In two other studies performed in pigs, (Goding et al 1988 and Marks1985), cutaneous blood flow measured by fluoroscopy was found to be reduced after inflation of expanders. However in both these studies measurements were made soon after inflation when the skin would be stretched tight over the expander and the tension within the expanders would be high. Not surprisingly when the expanders were deflated or removed the blood flow increased again. Blood flow in these circumstances will be so dependent on the tension within the tissues at the time of measurement that it is not a good indication of whether tissue expansion has any effect on skin blood flow in the long run. To determine this flow measurements have to be made several days after inflation when the has had time to adapt over the enlarged volume of the expander.

3.4 SUMMARY

Before a tissue expander is inflated beneath a skin flap several pathophysiological changes will have taken place. At the initial operation to insert the expander a subcutaneous pocket has to be made. This involves dividing blood vessels at the site of the incision and deep branches which enter the deep surface of the flap. It has been known for a long time that elevation of a flap prior to its transfer will enhance its blood supply, this technique being known as a delay. Several theories concerning the nature of vascular enhancement during delay are discussed but there is at present no conclusive explanation. In practical terms however undermining of a flap and incision of one margin which is normally performed on insertion of a tissue expander is seen to be one of the more potent forms of delay.

Although said to produce little reaction when implanted in animal tissues silicone elastomers have been shown to produce a very characteristic reaction. Considerable information has been gained from studies of the effect of breast prostheses which are enclosed by an envelope of similar material to that of the expander wall. A very definite capsule forms around such implants which is comprised mainly of collagen but also some capillaries it has a capacity to contract but has not been shown to have any beneficial effect on the vascularity of the overlying skin.

The effect of the above factors have not been studied in this project which is limited to the effect of the hydraulic distension itself. The response of skin to considerable forces of distraction is to elongate by a process of collagen fibre reorientation. If the forces become too great blood vessels supplying the skin will become obstructed and the skin become ischaemic. In Tissue Expansion it appears that some fibre reorientation occurs and the dermis thins as the collagen becomes more spread out however the evidence that there is a generation of new tissue by migration is inconclusive.

Some studies have been performed in order to investigate the effect of expansion on skin blood supply. It seems clear that there is a change in the appearance of small blood vessels but it is less clear whether there is an associated increase in blood flow. It is the purpose of the present study to determine this.

CHAPTER 4 THE EXPERIMENTAL MODEL

4.1.THE PIG AS A MODEL FOR SKIN FLAP RESEARCH.

Although early research into skin flap survival was performed on a variety of animals including the rabbit, (Webster J.P.1959), as the importance of the vascular anatomy of skin flaps was appreciated, (McGregor, I.A., and Morgan, G. 1973) it became important to use animals which have a comparable cutaneous blood supply to the human. Loose skinned animals differ markedly in this respect as the have a well defined panniculus carnosus on the deep surface of which is an extensive vascular plexus not present in man. In the pig however this muscular layer is poorly developed, (Montagna and Yun 1964), and the pig is considered by most investigators to have the skin which resembles that of man closer than any other experimental animal including other primates, (Donovan 1975, Langrana 1983 and Bustad 1965).

There are several difficulties with the pig. The animal is prone to malignant hyperpyrexia under anaesthesia and this appears to be an inherited condition. Temperatures of the animals were measured under anaesthesia but fortunately this problem was not encountered during this study.

Secondly there are problems using this animal for projects of more than a few weeks duration due to the animal's phenomenal growth rate. Although it is feasible to start work with a piglet weighing 20 - 25 kgs. the animal may grow to over 500 kgs. within a year. As flap

dimension will be significantly increased by growth control and experimental flaps should wherever possible be within the same animal. The pig at the best of time is a boisterous animal and induction of anaesthesia may be a sporting time. This problem worsens considerably as the pig grows.

Fortunately the pig also appears to be an excellent model for Tissue Expansion,(Cherry et al 1983, Sasaki and Pang 1984, Marks et al 1985 and Vanderkolk et al 1988) and this is due to the fact that it has relatively fixed skin. Major problems exist in trying to expand skin flaps in loose skinned, lagomorph, animals. To generate sufficient tension in these animals very large volumes have to be injected because there is considerable "Recruitment" of adjacent skin over the expander and thus there must be doubt about how much can be extrapolated to the human situation.

4.2. AIMS

It was the aim of the first part of the project to review the pig buttock flap, determine the anatomy of its blood supply and to evaluate it as a suitable model for tissue expansion for use in this project.

4.3.THE PIG BUTTOCK FLAP

4.3.1.Anatomy

In this study the pig buttock flap as described by

Daniel and Kerrigan in 1979 was used. This is a large flap over the buttock in an area of skin largely devoid of panniculus carnosus. The skin in this region has three sources of blood supply; random cutaneous vessels entering all round the periphery, musculocutaneous perforating vessels entering from the buttock muscles on the deep surface of the flap and finally a large direct cutaneous vessel, (the deep circumflex iliac artery). The flap is designed by first palpating the anterior superior iliac spine and the skin overlying this bony prominence forms the anterior superior corner of the flap. It is recommended that a flap 18cms. long and 10cms. in breadth is created.

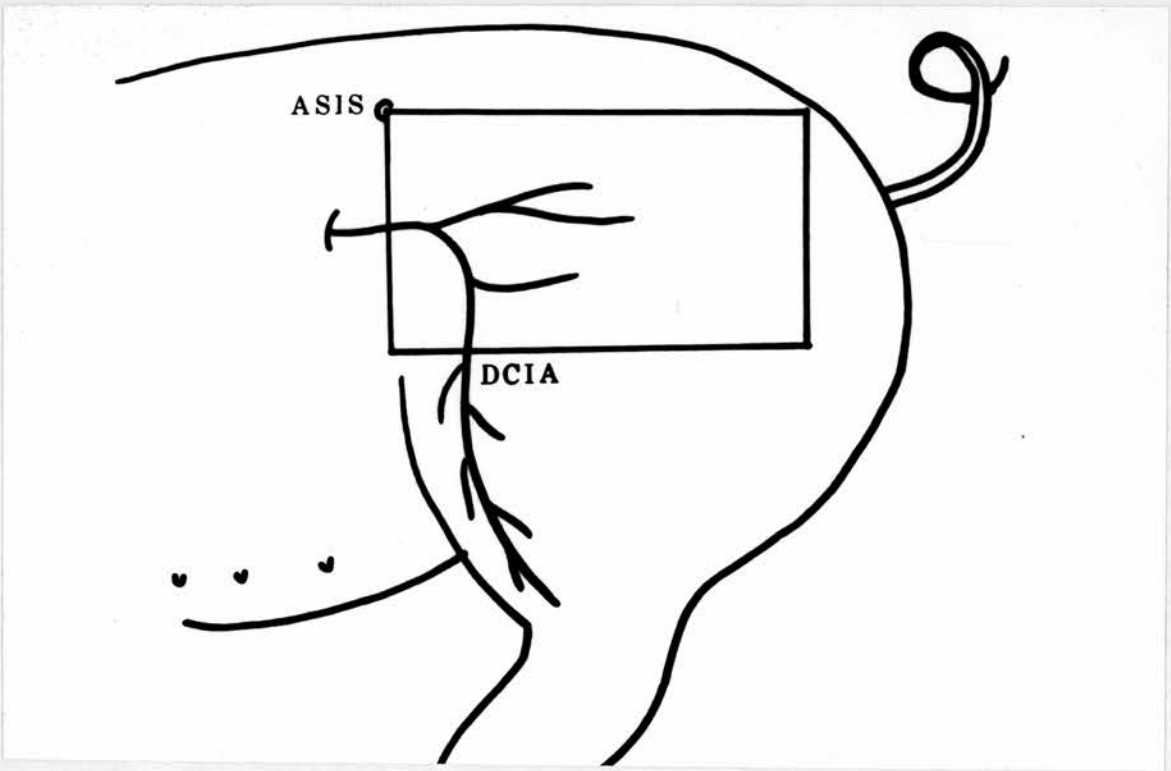


Figure 4.1. Diagram of Pig Buttock Flap showing course of deep circumflex iliac artery, (DCIA).

The deep circumflex iliac artery is a branch of the external iliac artery and originates within the abdomen. After passing through the abdominal wall and giving off muscular branches it appears superficially in the groin as a purely cutaneous artery anterior to the buttock at a point halfway along the anterior border of the flap, (Figure 4.1).

During its course the artery and its accompanying venae commitantes are joined by the lateral femoral cutaneous nerve. The cutaneous distribution of this nerve has been mapped out electrophysiologically, (Daniel et al 1975) and it is possible to raise this flap maintaining its innervation.

In order to confirm the course of this artery and define its territory of supply three pig carcasses were obtained for dissection. The vessel was identified within the abdomen and cannulated to enable injection of indian ink. Staining appeared not only in the skin of the pig's buttock but also along the anterior surface of the animal's hind leg, (Figure 4.2).

Dissection confirmed that although the artery did supply most of the buttock it was not an end artery which gave off its final branches into the flap as suggested by Daniel. Instead, after giving off large branches which pass caudally along the buttock flap, the vessel crossed the anterior part of the flap to pass down the anterior surface of the hind leg supplying the overlying skin. Thus when the flap is raised it is necessary to divide the vessel as it crosses the inferior margin of the flap.

Figure 4.2 Ink injection study of deep circumflex iliac artery showing perfusion of flap and hind leg.

These anatomical arrangements were found to be consistent in both varieties of pig used in this study.

4.3.2.Variants of Flap

Depending on how the flap is raised several different types of flap may be produced. If a proximally based flap is raised in a standard fashion it will produce an axial pattern flap, based on the deep circumflex iliac artery. This can be converted to an island flap by dividing all the flap's proximal attachment except the vascular pedicle. This pedicle may be traced proximally to the inguinal ligament where it may be divided to produce a free flap with a pedicle length of some 10 to 12 cms. in length. The vessel

calibre varies from approximately 1 to 2 cms. depending on the size of pig, which makes it suitable for a microvascular anastomosis.

Random pattern flaps can be created by basing the flap either proximally or distally however for a proximally based random pattern flap the neurovascular bundle, which is easily identified, must be divided.
In Daniel's original description of the buttock flap the following mean surviving lengths were found for each type of flap, (in centimetres).

Flap type	n.	Mean Surviving Length	Range
Arterial	7	13.3	11.4-15.6
Prox.Based Random	7	8.3	6.4-10.3
Dist.Based Random	6	4.6	3.0-7.2
Island	5	13.2	11.5-17.5
Free	5	13.5	10.5-17.5

4.2.3.Suitability of the Experimental Model for Tissue Expansion

A pilot study was performed on one pig to evaluate the suitablility of the flap for tissue expansion. Under general anaesthesia a subcutaneous pocket was created by incising the dorsal border of the flap and undermining the whole flap at a level deep to the subcutaneous fat. An expander was inserted which, when empty and inserted flat, occupied most of the pocket. This procedure was repeated on the other buttock and after recovery of the pig one expander only was serially inflated over a period of 8 weeks and the other was left empty as a control. In this manner it was possible to examine the effects of expansion alone, excluding any effect of flap delay that might have resulted from the initial insertion of the expander.

It was possible to inject approximately 100 ml. a week and by 8 weeks a total of 700 ml. had been inflated without significant difficulty resulting in marked expansion of the experimental buttock of the pig.

4.4.EXPERIMENTAL PROCEDURE.

The effect of Tissue Expansion on cutaneous vasculature was investigated by using the following procedure. A total of fourteen pigs were used; the first animal formed the above pilot study to ensure that the model would work satisfactorily. In one further pig an expander extruded due to infection thus necessitating

that the pig was destroyed and eliminated from the experimental series. The twelve remaining pigs were divided into two groups of 6. The first group, (series 1), were comprised of Yuccatan Gottinger piglets. This is a variety of miniature pig which was chosen not only to reduce difficulty in controlling larger animals but also in order that their reduced growth might have less effect on flap dimensions during the period of expansion. These pigs proved not only expensive but difficult to obtain and so common Landrace cross piglets were used in the second half of the study, (series 2).

4.4.1. Insertion of the tissue expander.

Having been fasted for six hours each animal was premedicated with an intramuscular injection of azaperone administered 20 minutes before operation. Anaesthesia was then induced and maintained on a mixture of 2% Halothane, 69% Oxygen, and 29% Nitrous Oxide, the animal breathing spontaneously through a mask.

In all pigs the hindquarters were shaved and both buttock flaps marked out with an indelible marker, (Figure 4.3). After full antiseptic skin preparation and draping the animal with sterile towels an incision was made along the dorsal border of the proposed flap and the whole flap completely undermined in the plane between fat and muscle by dissection with scissors, (Figure 4.4). After haemostasis was obtained with bipolar diathermy a remote valve tissue expander was placed under the flap and a subcutaneous tunnel

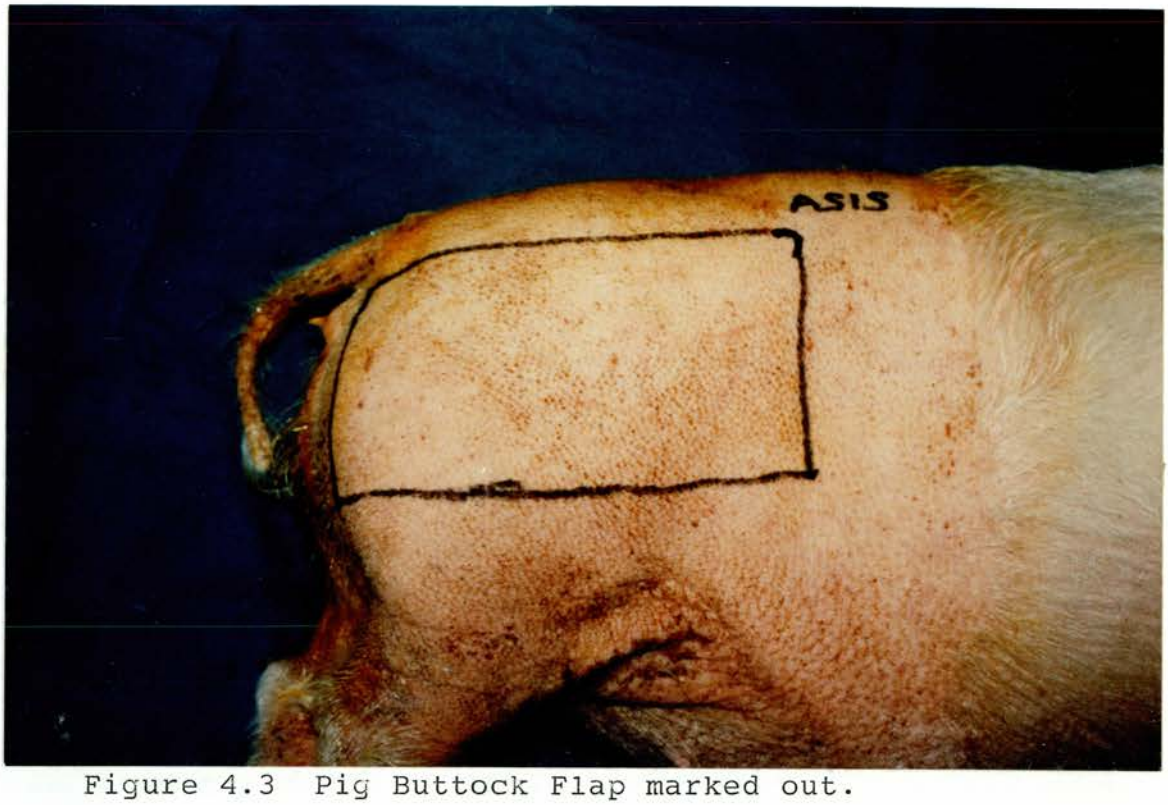


Figure 4.3 Pig Buttock Flap marked out.

Figure 4.4 Creation of subcutaneous pocket.

made dorsally towards the spine for placement of the valve. The wound was closed in one layer with an absorbable monofilament suture and the margins of the flap were then tattooed into the skin with carbon colloid. This was achieved by scoring the epidermis of the flap along the remaining three borders and applying tattoo ink over all four borders of the flap with a roll-on, (Figure 4.5).

Figure 4.5 Tattooing of flap margins.

The pig was then turned onto its other side and the whole procedure repeated on the other buttock flap. After both wounds had been sutured but before the animal was recovered the expander designated to be the experimental

side was tested to ensure fluid could be injected and aspirated freely. Approximately 100 - 200ml. of normal saline was then injected into the expander until it felt firm but not enough to exert undue tension on the wound. No drains were used, the wounds were not formally dressed but were covered with "Opsite" wound spray. Approximately 500 mls. of normal saline was administered intravenously and penicillin was given which was continued for 5 post-operative days orally.

Like some other experimental animals pigs may try to mutilate operation sites if they are a source of irritation and thus the decision not to use dressings or drains was made on the basis that the animals would probably have removed both when fully awake. No problem was encountered from not using a dressing but without drainage it was common for a seroma to develop around the expander. These were absorbed over a period of two or three weeks and did not give rise to any further problem.

Although before surgery the were housed together as soon as expanders were implanted the animals were kept in separate pens from which all movable objects on which the animals could scratch themselves had been removed. Some pigs did rub their buttocks on the bars but this caused minor scratches only.

4.4.2. Inflation of Tissue Expander.

Of the pigs numbered 1 to 12 those with even numbers had the left buttock expander inflated and the right

buttock expander left empty as a control but in those animals with odd numbers this pattern was reversed making the right buttock the experimental side. Post-operatively one week was allowed to elapse for wound healing to proceed before further inflation. the wound was then inspected and serial injections made at intervals of 4 - 7 days. In the first series of pigs an intramuscular dose of azaperone administered 20 minutes prior to inflation produced sufficient sedation for this procedure but in the second series the pigs required full general anaesthesia to permit the expanders to be inflated. This was administered as given at the initial surgery.

On each occasion a 23 gauge "butterfly" needle was used to puncture the subcutaneous valve after adequate skin preparation. The "butterfly" was connected by way of a three way tap both to a sphygomomanometer to enable measurement of pressure within the expander lumen and to a syringe to enable injection of saline into the expander. Between 50 and 200 ml. were injected on each occasion until the expander felt firm. The pressure immediately before inflation and immediately after inflation was measured and recorded, (Appendix 1). The range of pre-inflation pressures was 2 - 15 mms. Hg and post-inflation 17 - 28 mms. Hg. The decision on how much fluid to inject was made solely on clinical grounds and on no occasion was so much fluid injected as to cause blanching of the skin. Consequently no problems were encountered from pressure necrosis. In both series the

recommended volumes of the devices were exceeded by a considerable margin as has been found permissible, (Hallock 1987). A mean period of 8 weeks was required to complete inflation, (Figure 4.6), the cumulative volumes for each animal being given in appendix 1.

Figure 4.6 Expansion complete with experimental flap distended and control flap empty.

In general the pigs tolerated the procedure well and no analgesia was given at the time of inflation as is usual in human subjects.

It was concluded that the pig buttock flap would make a suitable model for this study.

CHAPTER 5 THE TERRITORY OF CUTANEOUS ARTERIES AFTER EXPANSION

The skin of an island flap is not only supplied by the axial vessel but also by the many random pattern vessels which enter the flap all round its periphery until such a time as that flap is raised on its pedicle. It follows that when such a flap is expanded the increase in surviving area which has already been described may be sustained by random vessels which may either may already exist or may be produced by the vascular ingrowth suggested by Cherry. Alternatively the territory of specific cutaneous vessels may enlarge to accommodate the increase in skin area, (Figure 5.1). By selecting an island flap for this project it was possible to raise the flap solely on a specific artery, (Deep circumflex iliac), and its venae commitans in order to enable a study to be made of this vessel's territory of supply as the overlying skin is expanded.

5.1 AIMS

The aim of this first part of the study was to determine how the territory of cutaneous vessels change as the overlying skin is subjected to expansion.

5.2. METHOD

The six pigs of series 1 were used for this study; They were aged approximately 8 weeks and had a mean

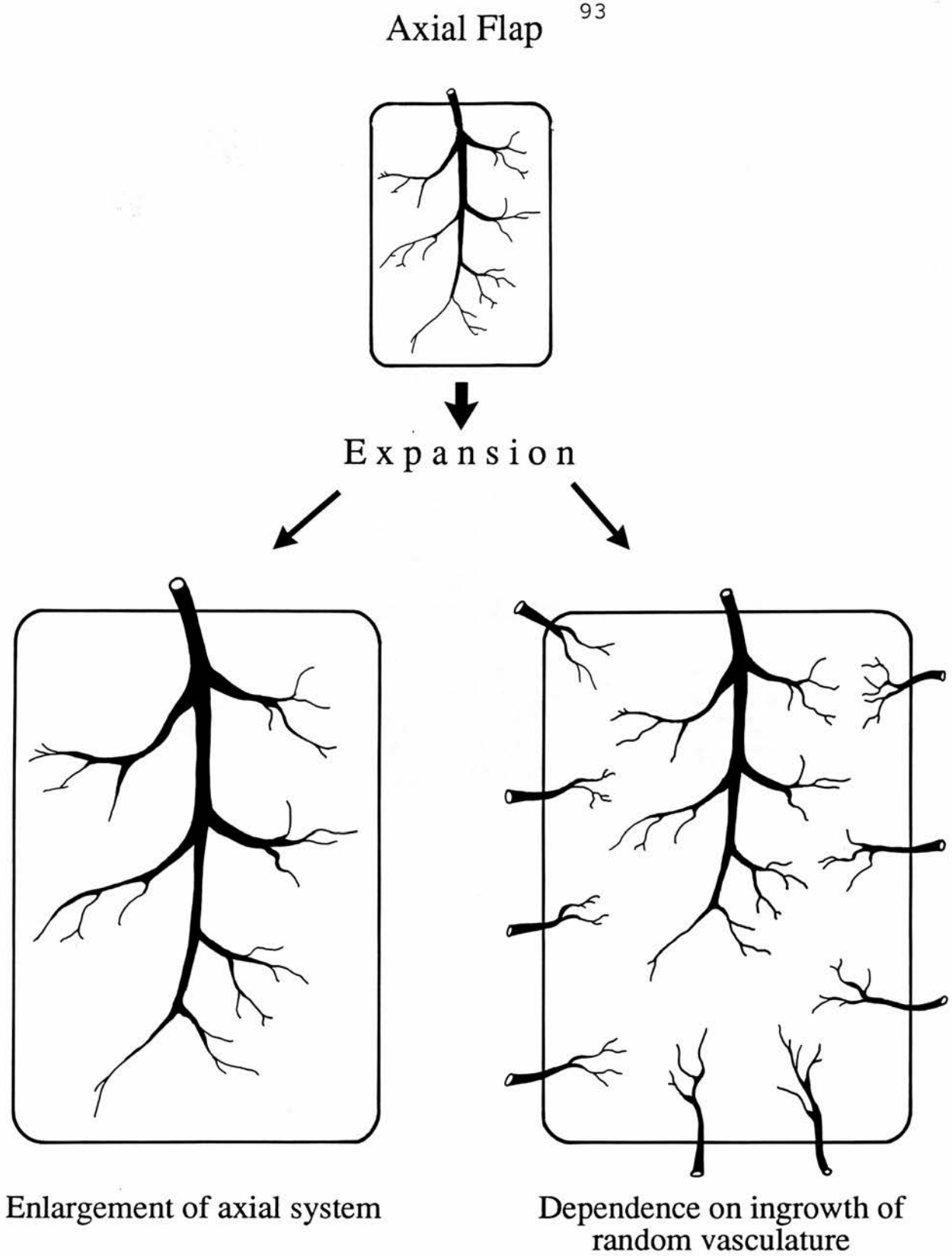


Figure 5.1 Possible mechanisms for the survival of skin following tissue expansion.

starting weight of 20 kg. These animals were piglets and some growth was therefore to be expected. In the first animal of this series the flaps marked out were 10 cm. in breadth and 17 cm. long; as will be seen there was almost complete survival of this pair of flaps and so in the remainder of the animals in this series the flap length was increased to 20 cm., the breadth remaining 10 cm.. Following the protocol previously described, after each flap had been completely undermined a 15 X 8 cm. rectangular expander was placed under each flap.

At weekly intervals the tissue expander on one side only was inflated untill it felt firm the other being left empty as a control. In the first animal a total of 750 ml. was injected into the experimental side but in order to to provide a greater challenge to the flaps in the remaining animals 1000 ml. was used.

Between 6 and 9 weeks were required to achieve tissue expansion to the volumes already predetermined. One week after this volume had been achieved each animal was then re-anaesthetised and prepared for surgery in a manner identical to that before. It was lain on the experimental side and the control flap was raised by first incising the dorsal, caudal and ventral borders of the flap. The expander capsule was soon identified and opened to enable the flap to be easily elevated proximally to the cephalad border until the anterior border of the hind leg muscles had been identified. At this point the skin only of this border was carefully incised leaving the flap

attached only by a strip of subcutaneous fat which included the axial vessels. As the fat was translucent the vessels could be identified and the fat was divided by careful dissection with fine scissors enabling the vascular pedicle to be cleaned of all tissues over a length of one centimetre where it entered the flap. Thus the flap was completely islanded, (Figure 5.3). Haemostasis was then achieved and leaving the empty expander in situ under the flap, the flap was returned to its bed and sutured back to the surrounding skin with a continuous absorbable monofilament stitch. The animal was now turned and the expanded flap was raised and islanded in an identical manner and, again leaving the expander in situ with the volume unaltered, the flap was sutured back in its bed. After spraying the wounds with a plastic dressing the animal was recovered from the anaesthetic.

Figure 5.3 Flap islanded on its vascular pedicle.

For one further week the animals were fed normally after which time any skin which had been rendered ischaemic as a result of islanding the flaps had undergone frank necrosis and a clear line of demarcation existed, (Figure 5.4).

Figure 5.4 Pig one week following islanding of both buttock flaps which show necrosis distally.

Each pig was then again premedicated and killed with an intracardiac injection of barbiturate to allow the following measurements to be made.

1 Each animal was weighed.

2 The area of the whole of each flap, both surviving

and necrotic was measured. As the shape of the expanded flap was very complex geometrically, being a large ovoid surrounded by a flat rim measurement was simplified by covering the flap with rows of adhesive tape of a known width of 1.3 cm. By multiplying this with the total length of tape needed to completely cover the flap it was possible to calculate the flap area with a high degree of accuracy.

3 Flap survival was quantified by a comparison of the surviving lengths of each flaps. Using a standard ruler graduated in millimetres the length of surviving and total lengths of the flap were made along the dorsal and ventral borders and in the midline of each flap. The mean of each set of three figures was taken to represent surviving and total flap lengths.

5.3. RESULTS

The weights of the pigs at the completion of the study are shown in table 5.1. The mean weight of the pigs was 25.7kg.. The increase in weight, after exclusion of the weight of the inflated expanders, was due to growth which also accounted for the increase in size of the control flaps.

The total flap areas for each flap are shown in Table 5.2. There was an average increase of 95% in the expanded flaps,(mean 485cm.^{2}) compared with the control flaps, (mean 249cm.^{2}).

The surviving and total flap lengths for each pig are shown in Table 5.3. The mean of the surviving lengths of the expanded flaps was 30.8 cms. and that of the control flaps was 21.8 cms.. This demonstrates an increase in surviving length of nearly 50 per cent following tissue expansion, and this increase was found to be significant when tested (paired t test; $p < 0.001$). The survivng flap length measured as a percentage of total flap length is shown in Table 5.4, with a mean of 95.7 percent of the expanded flaps surviving and 96.7 percent of the control flaps, which was not found to be a significant difference.

TABLE 5.1 **PIG WEIGHTS**(kilograms)

Pig	Starting Weight	Final Weight*
1	19.0	26.5
2	19.0	25.0
3	19.5	26.5
4	21.0	31.0
5	22.0	23.5
6	19.5	21.5
mean	18.2	25.7

*The weight of the full expander has been deducted.

TABLE 5.2 **COMPARISON OF FLAP AREAS**

Area of flap surviving measured in $cm.^2$

Pig	Expanded Flap	Control Flap	percentage increase
1	388	228	70%
2	493	240	105%
3	480	246	95%
4	528	270	96%
5	511	251	104%
6	512	261	96%
mean	485	249	95%

SURVIVAL OF ISLAND FLAPS

TABLE 5.3 Surviving and Total Flap Lengths

cms.

	Expanded Flap		Control Flap	
Pig	Surviving	(Total)	Surviving	(Total)
1	25.5	(26.5)	18.5	(19.0)
2	31.0	(34.0)	23.0	(24.0)
3	29.0	(31.0)	20.0	(22.5)
4	36.0	(39.0)	23.0	(24.0)
5	31.0	(31.0)	23.0	(23.0)
6	32.0	(32.0)	23.0	(23.0)
mean	30.8	(32.3)	21.8	(22.6)

TABLE 5.4. Surviving Flap Length Measured as a Percentage of Total Flap Length

Pig	Expanded Flap	Control Flap
1	97%	99%
2	91%	96%
3	94%	89%
4	92%	96%
5	100%	100%
6	100%	100%
mean	95.7%	96.7%

5.4. DISCUSSION

The survival lengths of the pig buttock flap raised acutely reported in Daniel's original description of the flap were deduced from studies on pigs of a similar weight to those in this series. Therefore although it would have been possible to have a third group of pigs who had flaps of similar dimensions raised acutely to compare survival of this type of flap it was considered that this would have contributed little to the study and thus been wasteful of animals.

It rapidly became apparent that the effect of delay by insertion of the tissue expanders enabled very much larger island flaps to be raised on the pig buttock. For this reason the length of the flaps had to be increased but the size of the pig's buttock limited the total flap length to approximately 20 cms. if a distance of at least 5 cm. was to be preserved between the distal end of the flap and the anus. This was long enough to provoke necrosis in all but two of the pigs. Although in Cherry's studies on survival of random flaps expansion was limited to 100 or 250 ml. it was felt that these volumes would not themselves generate a sufficient increase in flap length for statistical difference surviving lengths to be shown.

At the second operation the control side was islanded first in each pig because by this stage the inflated expander had caused a considerable swelling in

the pig's hind quarters and if the full weight of the animal was allowed to lie on this side after its flap had recently been islanded, (during the control flap elevation), some circulatory embarrassment could have arisen in the compressed flap.

The expanders were left in the pig at this stage to maintain the expanded flap's tension and thus prevent skin contracture. In a project involving a similar surgical procedure,(McCann 1988), it was intended to prevent this from happening by excision of surrounding skin and suturing the expanded flap into a larger skin defect, however as the experimental flap was by this time very nearly hemispherical it was not possible to stretch it out into a two dimensional plane. In addition the presence of the expanders prevented any beneficial effect which may have resulted from an increased vascularity of the beds from affecting flap survival.

Flap length was used to quantify flap survival as the breadth of the flap was not a critical factor in flap survival. Flap area was measured however not for comparison of survival but as an indicator of the degree of expansion that had been achieved in the experimental flap.

5.5 SUMMARY

Growth occurred during the two months of the experiment and resulted in an average weight gain of 5.7kg. and an increase in area of 49 $cm.^2$ of the nonexpanded control flap.

Following expansion however the area of surviving flap was increased by a factor of 49% although this did not correspond to the maximum area that could be sustained by the axial vessel. The maximum surviving flap length which can be sustained by the axial vessel was increased from a mean of 21.8 cm to a mean of 30.8 cm. after expansion, an increase of nearly 50%. The proportion of flap survival to flap necrosis however appeared to be unchanged.

CHAPTER 6 BLOOD FLOW IN EXPANDED SKIN

In Chapter 5 an increase in the surviving length of skin flap that could be sustained by a single vessel after tissue expansion was shown. This could have been achieved by an improved distribution of blood supply within the expanded flap either with or without an increase in total blood flow into the tissues. If no increase in blood flow took place as the larger area of skin would be still supplied by the same volume of blood it would suggest that expanded skin does not have the increased vascularity claimed, particularly if there was a generation of new tissue and an increase in tissue bulk of the flap.

6.1 AIMS

The main purpose of the second part of the project was to measure the blood flow proximally in the deep circumflex iliac arteries in a second series of pigs, which had undergone a similar surgical procedure.

The flap temperatures were to be measured and compared as a further indication of any difference in blood flow that there might be between the two sides.

In addition to measurement of total blood flow by measuring the weight of each flap it would be possible to derive the blood flow through a unit weight of tissue.

6.2. METHOD

The animals of the second series were prepared for theatre in a similar manner to the first. Under general

anaesthetic flaps were marked out on each buttock 10 cm. square and through a dorsal incision the whole flap was undermined to permit insertion of a round tissue expander* 10cm. in diameter (designed to be inflated to 1000 ml.). No drains were used but antibiotics were given as before. Post-operatively one side only was chosen for expansion and over a period of some 8 to 10 weeks this was inflated to 1000 ml.. Individual expansion volumes are given in the appendix. Each animal was then returned to theatre for blood flow measurements to be made. Although for each individual pig any factors which might cause a change in skin blood flow such as blood oxygen saturation and temperature would affect either side in a similar manner great care was taken to ensure measurements were made in all pigs in as near as possible identical physiological conditions. Thus for this part of the experiment endotracheal intubation was performed to reduce the risk of hypoxia and hypercapnia which might increase cutaneous blood flow. Arterial blood gas samples were taken during anaesthesia and analysed to ensure normal conditions. Room temperature was recorded and a thermometre inserted rectally to enable measurement to be made of the pig's core temperature during blood flow measurements in each flap.

In order to prevent hypovolaemia normal saline was infused intravenously into each animal at the rate of 500 ml. per hour.

When the animal was stable under anaesthetic an

incision was made along the anterior border of the expanded flap. A further horizontal incision was made anteriorly half way along this incision to allow two triangular skin flaps to be raised and turned forwards to give access to the subcutaneous tissues anterior to the flap where the vascular pedicle lay. By very careful dissection the deep circumflex iliac artery was identified and carefully cleaned of all adventia over a length of 2 cm. just proximal to its entry into the flap, (Figure 6.1).

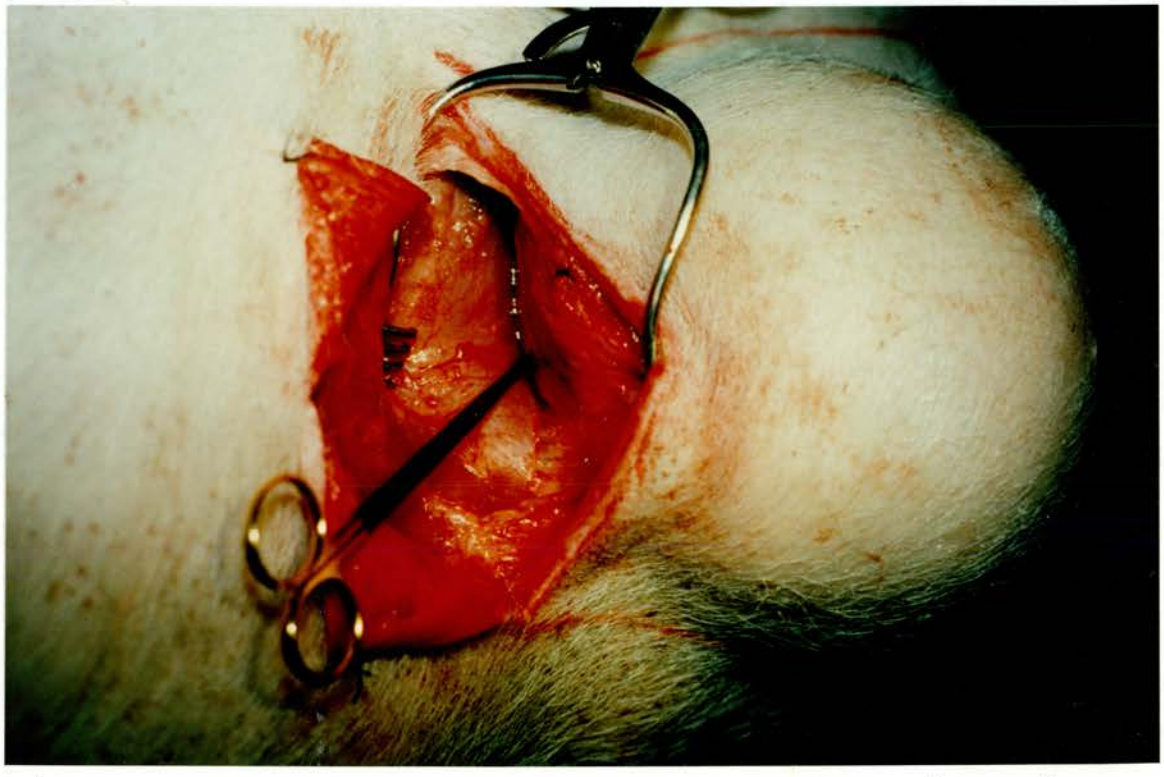


Figure 6.1 Exposure of flap vascular pedicle, (at tip of scissors).

To overcome any spasm that might have occurred as a result of surgical manipulation or cooling due to exposure maximal pharmacological dilatation was achieved

by dropping 5 ml. of a solution of 1% plain lignocaine over the vessels and returning the triangular skin flaps over the wounds to allow the tissues to warm up for 5 minutes. During this period a 5 mm. stab incision was made in the centre of the skin flap and a thermocouple probe inserted through this into the subcutaneous tissues.

Core temperature of the pig was then measured, the room temperature was also noted and the temperature of the superficial layers of the flap recorded using the thermocouple. The skin flaps were then retracted again and blood flow through the artery was measured with an electromagnetic flowmeter, (Gould Statham SP 2202), (Figure 6.2). A C shaped flow probe of 2mm. internal diameter was placed around the artery and at right angles to it where the vessel had been dissected clean. This provided a snug fit between the artery and probe without causing stenosis, (Figure 6.3). The ground ring of the flow probe was sutured into nearby subcutaneous tissue. The electromagnetic flowmeter was connected to a Devices recorder,(Model M2), which could be calibrated at a full range of either 0-30 ml./min. or for higher flows at 0-100 ml./min., (Figure 6.4).

Recommended procedures previously published were followed in order to prevent artefactual inaccuracy from occurring during flow measurements, (Cannon et al, 1962, Gordon et al, 1971, Harper et al, 1970, Vance et al, 1979, Banis et al, 1980, Nigra et al, 1981, McKee et al, 1982).

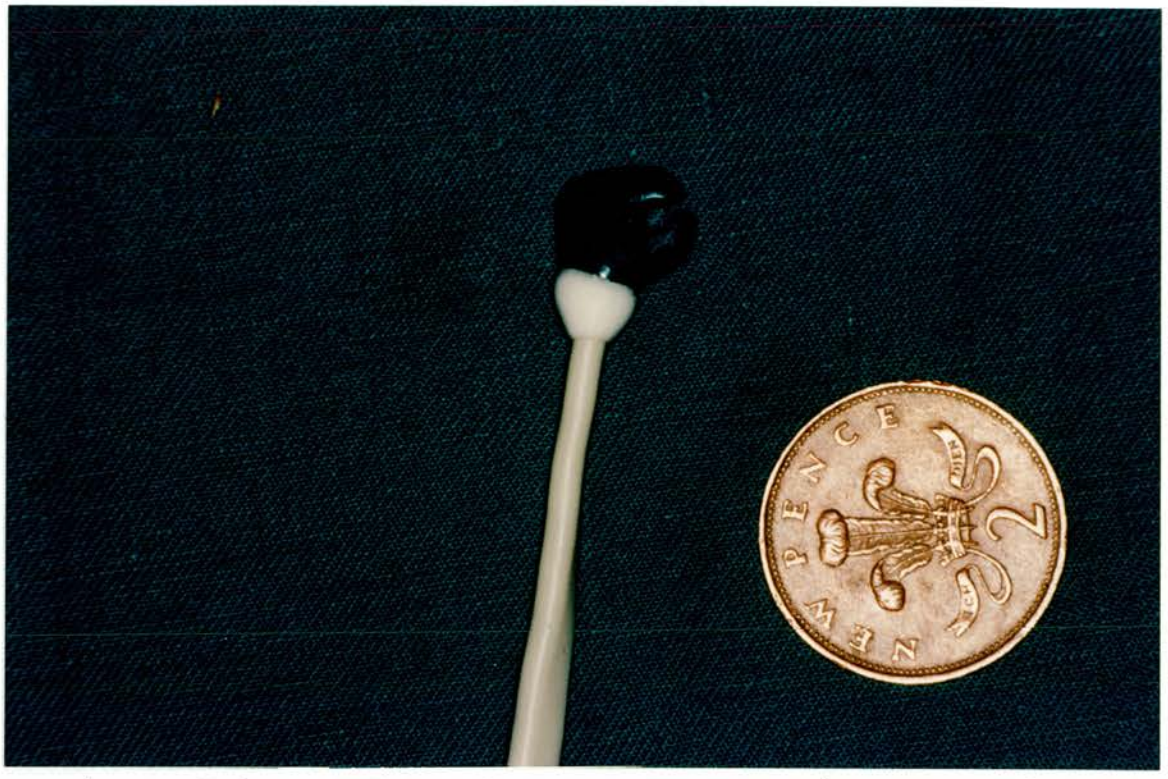


Figure 6.2 Gould Statham electromagnetic flow probe

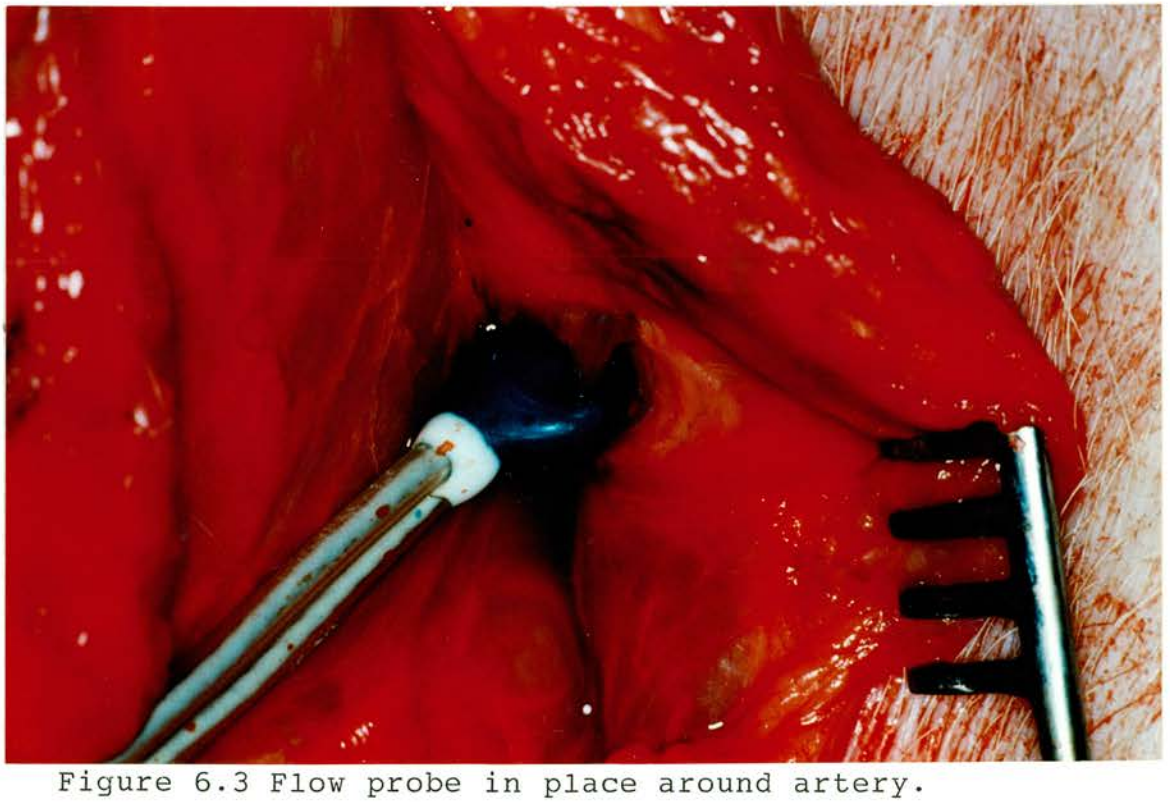


Figure 6.3 Flow probe in place around artery.

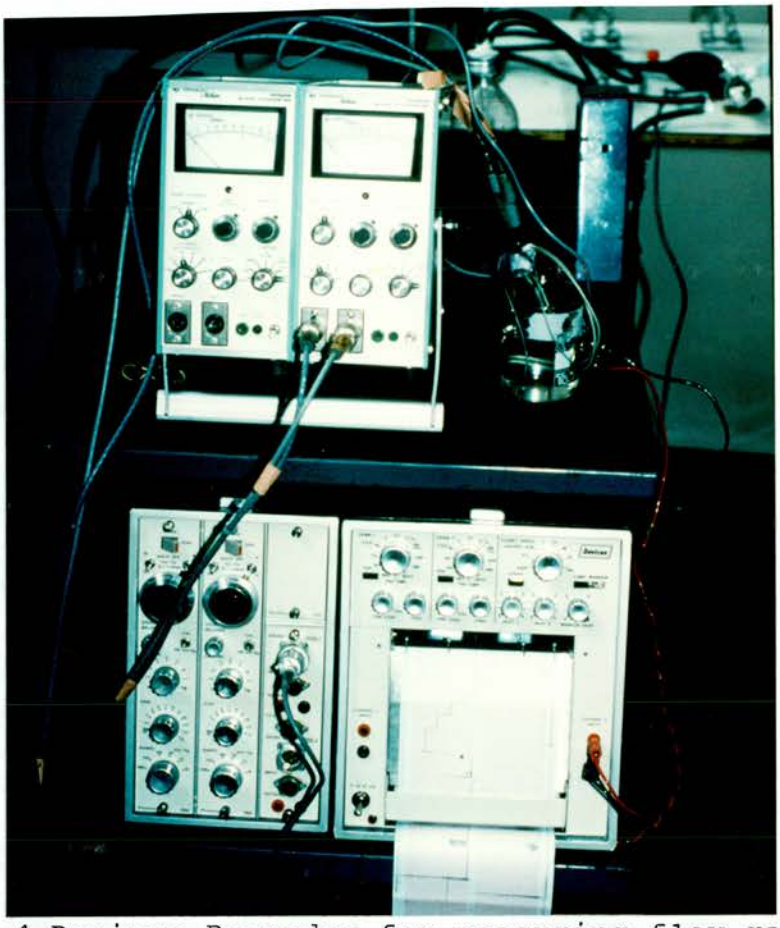


Figure 6.4 Devices Recorder for measuring flow rates.

After an initial depression of blood flow as a result of application of the probe the blood flow was seen to steadily increase until, within 2-3 minutes, a constant level was reached and then recordings were made.

The pig was then turned and exactly these same procedure performed to measure blood flow and temperature in the control flap. The pig was then destroyed by an intravenous injection of barbiturate and in order to determine whether there was any increase in tissue mass as a result of tissue expansion a square of skin which was centered on the middle of the expander 18 cm. by 18 cm.

was excised including all deep tissue down to muscle. The expander was removed and the pair of flaps weighed. A square larger than that expanded was excised in order to include a margin of skin from which recruitment might have occurred as this might have been a cause for an apparent increase in flap weight.

The results of the expanded and control sides were then compared using a paired t test. Total blood flow, flap temperature and blood flow per 100 gm. tissue values were subjected to the test.

6.3 RESULTS

When the inflated expander was removed from pig no.8 a considerable volume of purulent fluid was unexpectedly found around the inflated expander. As a coliform was found in large numbers in this fluid this pig was deemed to be infected and excluded from comparison.

There was no significant difference in flow rates between expanded and control sides. The mean rate for the expanded flaps being 24.6ml/min. and the control flaps 24.8ml/min. The individual results are given in Table 6.1 and a typical section of the flow tracings, (for pig no.10), are shown in Figure 6.6.

The temperatures of the flaps and environment are given in Table 6.2. It will be seen that in all but two pigs, (one of which had the infected expander), the temperature of the control flap was higher than the corresponding expanded flap; the mean temperature of the control being 36.0 degrees and that of the expanded 35.7 degrees. However this was not statistically significant when tested.

The weight of the flaps excised post mortem are shown in Table 6.3 and from these figures blood flow rates per 100 gms. of tissue have been deduced, (Table 6.4).

The areas of the flaps are given in Table 6.5 and as in chapter 5 there is a increase in the size of the expanded flap compared to the control; the mean increase being 52%.

TABLE 6.1 **AXIAL VESSEL BLOOD FLOW**

(mls./min.)

Pig	Expanded Flap	Control Flap
7	16	16
9	29	31
10	32	32
11	18	16
12	28	29
mean	24.6	24.8
8*	120	48

(* infected)

TABLE 6.2 **FLAP TEMPERATURES**

Degrees Centigrade

Pig	Expanded Flap	Control Flap	Core Temp.	Room Temp.
7	35.7	35.8	38.2	21.60
9	35.2	36.1	38.5	22.25
10	36.1	36.0	38.7	20.00
11	35.5	35.8	39.0	21.50
12	36.0	36.2	37.8	21.00
mean	35.7	36.0	38.4	21.30
8	37.1	36.7	39.1	22.75

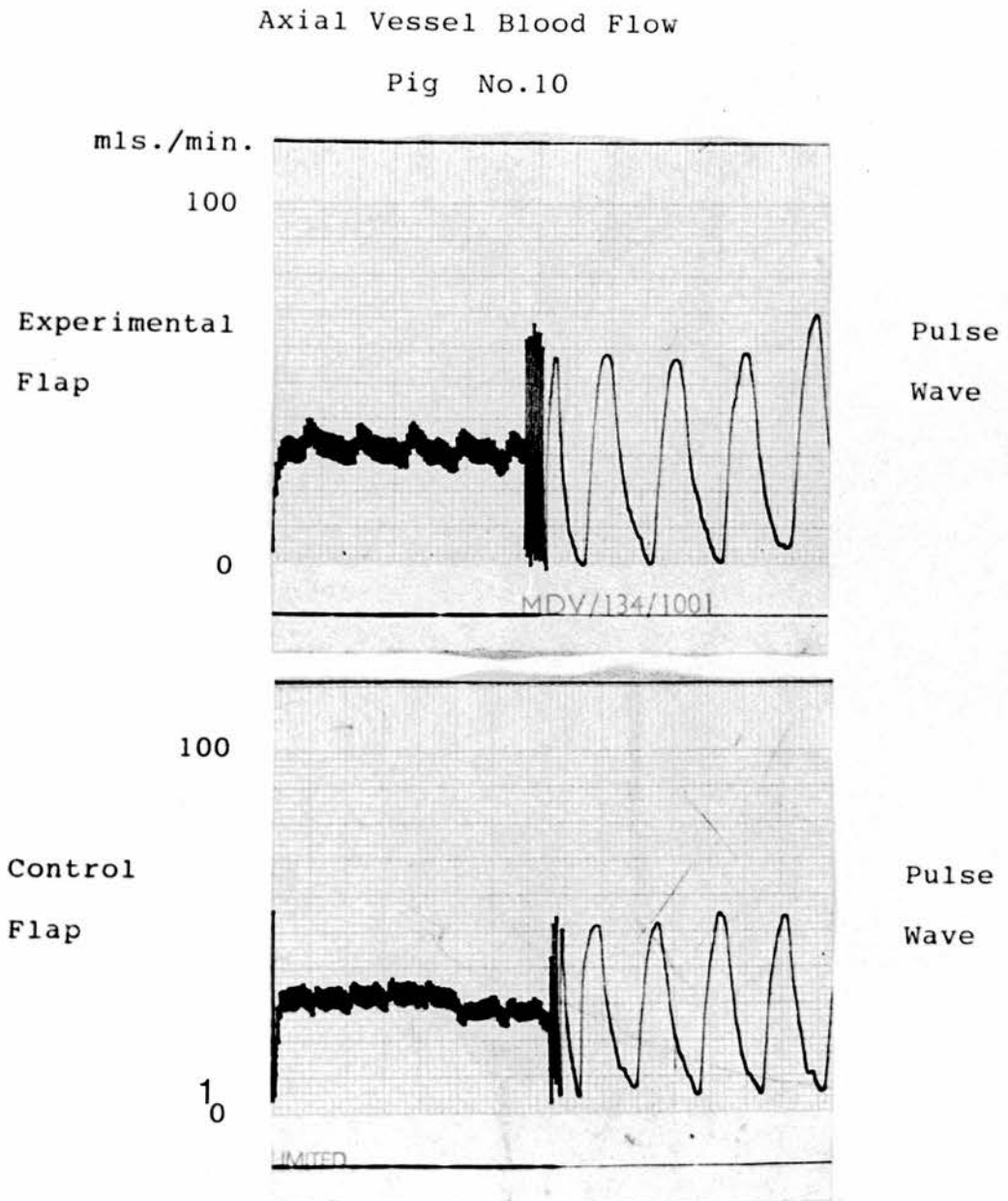

Axial Vessel Blood Flow
Pig No.10
mls./min.
100
Experimental
Flap
0
Pulse
Wave
MDV/134/1001
100
Control
Flap
0
Pulse
Wave
LIMITED

figure 6.6

TABLE 6.3 **FLAP WEIGHTS**

18 cm. Square Flaps (grams)

Pig	Experimental	Control
7	433	284
9	386	260
10	288	168
11	441	245
12	409	278
mean	391	247
8*	522	273

(*infected)

TABLE 6.4 **BLOOD FLOW per 100 gms. OF TISSUE**

(mls./min.)

(mls./min.)

Pig	Expanded	Control
7	3.7	5.6
9	7.5	12.0
10	11.1	19.0
11	2.5	6.5
12	6.8	10.4
mean	6.32	10.7
8*	23.0	17.6

TABLE 6.5 **FLAP AREAS**

Area of Flap 18cms. square

cms^2.

Pig	Experimental	Control	%increase
7	528	338	56%
9	487	333	46%
10	508	324	57%
11	430	341	26%
12	615	351	75%
mean	514	338	32%
8*	589	326	80%

6.4. DISCUSSION

There are several well tried methods of blood flow measurement. A method was required which could be applied to a single blood vessel and monitor over a period of time so that the flow could be observed to equilibrate after a period of time. This precluded the use of radioisotope clearance and microsphere studies. Of the remaining methods it was thought that Doppler flowmetry and plethysmography would not be as accurate and more difficult to use compared with an electromagnetic flowmeter.

Within the electromagnetic flow probe is an electromagnet which, receiving an electric current, sets up a magnetic field across the vessel placed within it.

As blood is an electrically conductive fluid it obeys Faraday's law of electromagnetic induction and an electric potential is generated at 90 degrees to both the magnetic field and the direction of blood flow, (Figure 6.5). The magnitude of this signal is measured and directly proportional to the blood flow, (Gordon 1971).

To ensure accuracy of this system strict attention to detail must be paid. There must be good contact between probe and vessel. This requires the vessel to be carefully cleared of all adventitia and a probe selected of a size which will snugly fit around the vessel without constriction. The probe has to be cleaned with pumice powder and soaked in Ringer lactate for 30 minutes prior to use according to recommended practice, (Banis 1980).

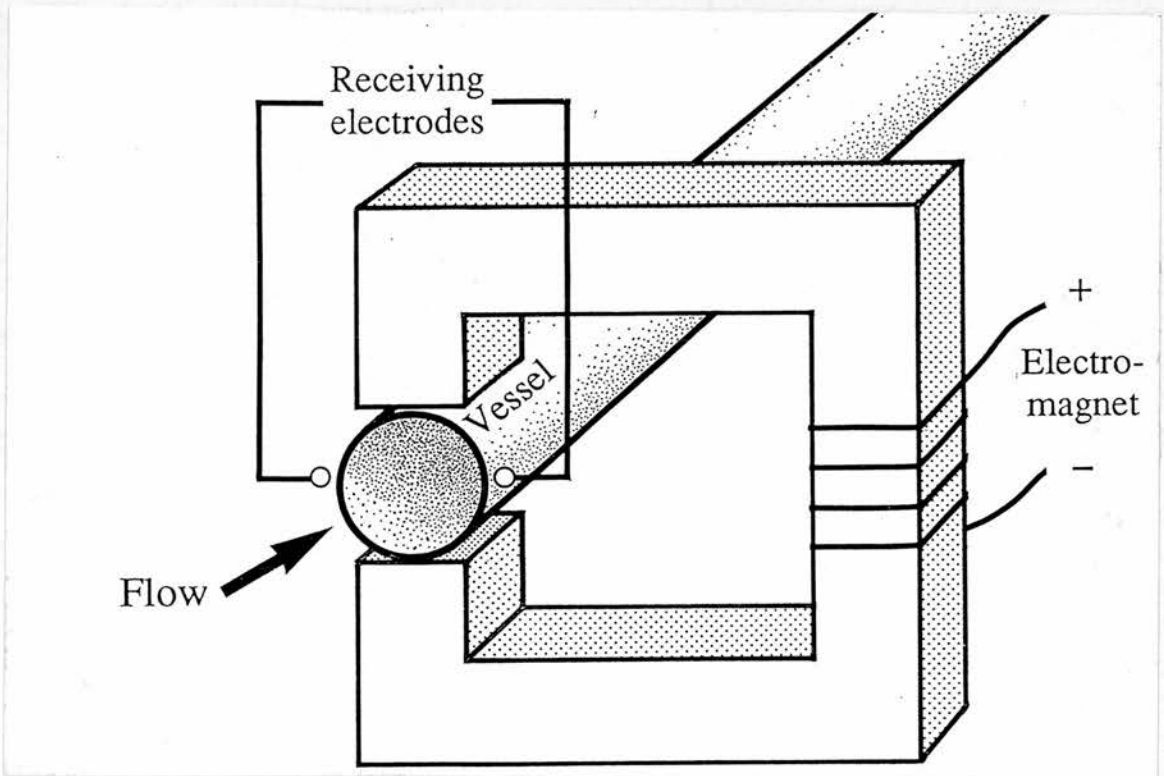


Figure 6.5 Principle of Electromagnetic Flowmetry

The second possible source of error with this method

is due to inaccurate calibration of the flow probe. During their manufacture the Statham flow probes are calibrated and standardised to an accuracy of within 5% at maximum recordings. The recording equipment must be adjusted according to the flow probe being used and an "electronic zero" set as a baseline. Prior to recording the baseline should be checked by making an "occlusive zero" by stopping blood flow temporarily with a clamp. The accuracy of this method of blood flow measurement has been established in the past and so was not investigated in this project. In two pigs flow rates were however checked by dividing the vessels immediately after measurements had been made and collecting the blood in a graduated cylinder against a stopwatch. The flow as measured by collection was within 15% of that recorded by flowmetry.

Measurement of skin temperature by needle thermocouple has long been recognised as a test for skin blood flow, (Brooks, 1925), and has in particular been used to study skin flaps, (Douglas and Buchholz 1943). Although it has been suggested that it is not specific enough to be reliable enough for clinical use, (Winsten et al, 1961), it is a very simple test to perform and, in the presence of a narrow range of both core and room temperatures, probably relevant. One unknown factor is the amount by which the presence of a litre bag of saline, separating the skin flap from the underlying bed, will reduce the heat transmitted out t the surface.

Flow rates through a unit weight of tissue, 100 gms., were deduced by dividing the measured flow rates by the weight of the 18 cm. square flap on the assumption that the vessel would supply the whole of this area. Although tissue bulk was deduced from the weight of the skin flaps excised postmortem in retrospect it would have been more accurate to have compared dry weights of tissue. This would have excluded any error that might have arisen from an increased in water content following tissue expansion. In practice however it will be seen that when the skin was analysed histologically neither flap was found to be particularly oedematous and thus it is considered that these comparisons are valid.

6.5. SUMMARY

No change in blood flow through the flaps axial vessel could be detected after tissue expansion using electromagnetic flowmetry. Further evidence supporting these findings were gained by the lack of any increase in skin temperature found in the expanded skin compared with the control skin.

Not only was the area of a flap 18 cm. square increased by a mean factor of 34% but the weight of such a flap was increased after tissue expansion by a mean of 144 gm.or 37%. Assuming this area of skin to be supplied only by the deep circumflex iliac artery this would mean that the rate of perfusion per unit of skin would fall following tissue expansion. The theoretical mean blood flow per 100 gm. of tissue being 6.32 mls./min compared to 10.7 mls./min in the control skin.

CHAPTER 7 VESSEL STRUCTURE

Since the increase in flap survival seemed not to be due to an increase in blood flow it can be concluded that it is due to a more effective distribution of flap blood supply distally in the flap. One way of achieving this would be by a change in the topography or architecture of the vascular tree.

In addition the structure of the blood vessels themselves would not only be an important factor governing perfusion of the overlying skin but be a consideration for microvascular transfer of such flaps. The free tissue transfer of pre-expanded free flaps would potentially be a major new reconstructive technique.

7.1. AIMS

The aim of this section of the project was to study changes in three aspects of vascular anatomy following Tissue Expansion:

1 Topography of the larger vessels of the skin flaps.

2 Histology of the smaller vessels within the skin flaps involving a detailed analysis of the number, location and size of vessels.

3 A study of the vascular pedicles proximal to the flap.

7.2. METHOD

Vessel Topography

The larger cutaneous vessels within the flap were studied by performing angiography in 5 pairs of flaps, two from pigs in series 1 and three from pigs in series 2. These were produced in the following manner; after slaughtering the pigs both flaps were excised at a plane between capsule and the muscle having previously dissected out a 2 cm. length of pedicle proximal to the flap. The deep circumflex iliac artery was then cannulated and an injection of an aqueous suspension of barium sulphate made and a radiograph was then taken. Vessels of a diameter of .25 mm. or more could be visualised. In the last three pairs of flaps the technique was standardised to 4 ml. of Barium Sulphate injected over 4 seconds and exposures were made 8 seconds after completion of injection.

Histology of small cutaneous vessels.

After completion of all other measurements 3 cm. square samples of skin was excised from the centre of each flap in series 2. The centre of the flap was the area which, in the experimental side, had undergone most expansion. The samples were pinned onto small blocks of cork to prevent shrinkage, as described by Southwood, (1953), and preserved in 10% formalin. The specimens were embedded in paraffin wax and two sections cut from each. One was stained with haematoxylin and eosin and the other

with Elastin Van Giesen stain in order to make blood vessels easily detectable.. Light microscopy was then used to detect any differences in architecture between the expanded and control samples.

Morphometry was performed on an instrument in the Department of Histopahology, Charing Cross and Westminster Hospital Medical School. The sections of skin stained for elastin were viewed by means of an inverted Leitz Laborlux microscope fitted with a fan cooled quartz halogen bulb. The image was reflected off a front-faced aluminised mirror and focused onto the optical coating of a transparent x,y co-ordinate digitiser tablet. This rear-projection technique thus allowed the complete image to be viewed from the front without obliteration by the operators arms, (Figure 7.1).

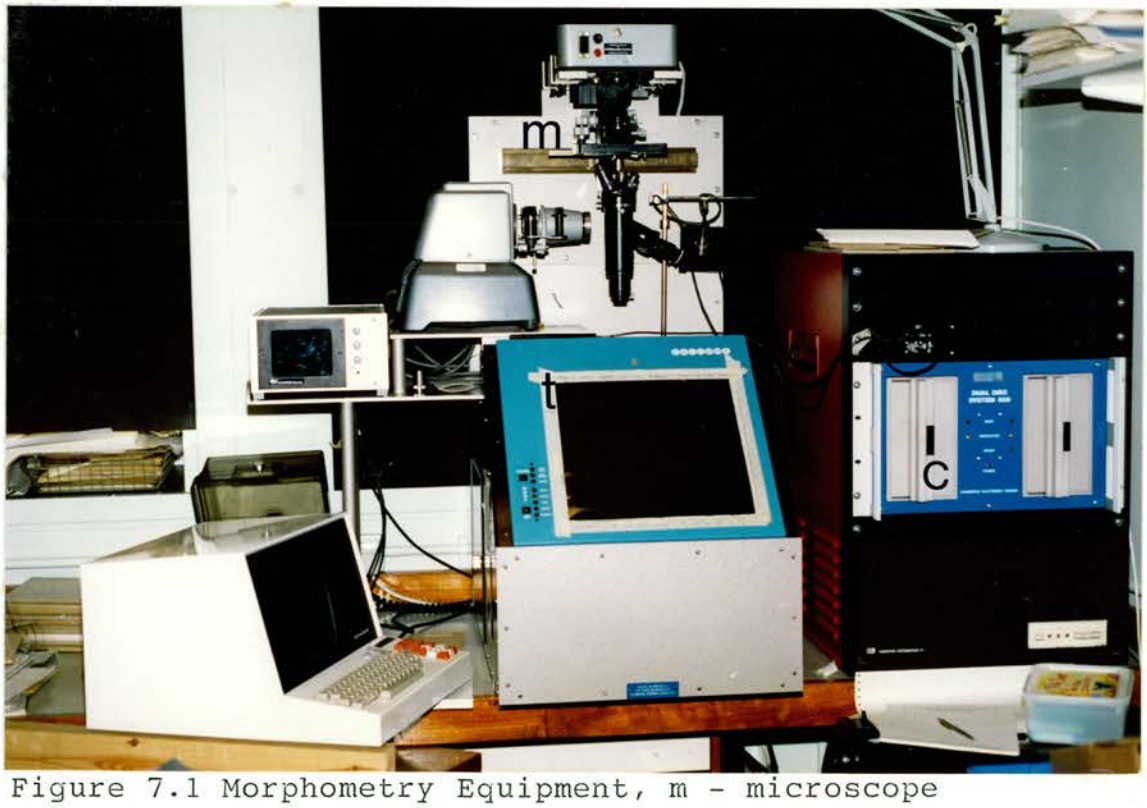


Figure 7.1 Morphometry Equipment, m - microscope

t - digitising tablet and c - computer.

The tablet operates by means of pulsed radio signals transmitted in turn through x and y planes by wires buried in the tablet. By means of a "pen" containing a radio receiver, tracing the image produces a signal in the pen which is related in time to the transmission signal and gives x,y co-ordinate transmission to a theoretical resolution of 0.1 mm. on the tablet surface. At the magnification used, the tablet surface area corresponded to about 1,500 microns of section in both x and y planes.

Signals from the co-ordinate digitiser were fed to a dedicated computer system, (Cambridge Electronic Design Ltd., Science Park, Cambridge, U.K.) with custom software to perform calculation from co-ordinate data to derive morphometric parameters. Thus the following structures were traced and their lengths/areas calculated.

i Epidermal thickness. The epidermal thickness was measured at 1 mm. intervals and a mean of 12 values taken as the epidermal thickness of that particular sample.

ii Dermal thickness was measured at 5 mm. intervals and the mean of 4 values taken.

iii The Fat Layer (from deep surface of the dermis to surface of the capsule), thickness was measured at 5 mm. intervals and the mean of 4 values taken.

iv Rate of undulation of the rete pegs was measured. This was achieved by measuring the length of the dermo - epidermal junction and dividing it by the length of the superficial surface of the epidermis, (Figure 7.2).

v The number and crossectional area of lumen of every blood vessel over 500 microns^2, (approximately 18 microns diameter), in the dermis, subcutaneous tissues and at the interface between the fat and capsule were measured.

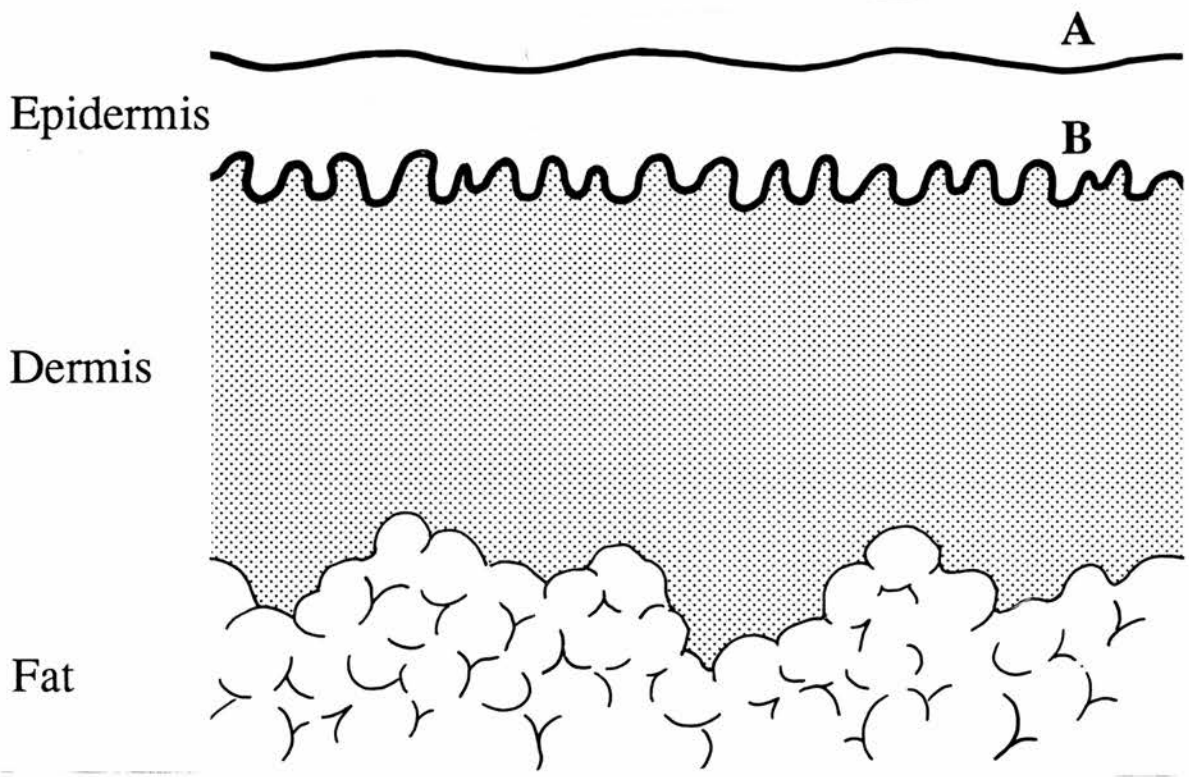


Figure 7.2 Rate of undulation of the epidermal dermal interface, taken to be the ratio of lengths A:B

The values obtained in measuring the thickness of the three layers, the vessel counts and the cross

sectional areas of the vessel lumens were all subjected to a paired t test to determine whether any statistically significant difference existed between the expanded and control sides.

Vascular Pedicles

In the second series of pigs the structure of the deep circumflex iliac artery in the vascular pedicles was also examined. Measurements were made of the external diameter of this vessel as it crossed the anterior border of the skin flap in vivo following full pharmacological dilatation at the time blood flow was investigated. Measurement was made with a set of Vernier calipers accurate to 0.05 mm.. The results were tested with a paired t test.

Following slaughter, and angiography if performed, a section of the pedicles, comprising of artery and two venae commitans were removed and preserved in 10% formalin. It was not possible to cut sections absolutely squarely across the vessels and so quantitative measurements of external and internal diameter could not be made with any degree of accuracy but a qualitative histological assessment was made of the vessels from experimental and control sides.

7.3 RESULTS

The first pair of angiograms produced showed some striking differences between expanded and control flaps, (Figure 7.3 and 7.4). Notably there appeared to be a considerable increase in the calibre of the axial vessel and a proliferation of the smaller vessels in the expanded flap. In the second pair of angiograms similar changes were seen in the axial vessels but the films were not of very good quality as they were partly obscured by dye spillage and no conclusions could be drawn concerning the smaller vessel network.

However three further pairs of angiograms were produced under identical conditions of dye injection and previous observations were not upheld. In none of the three pairs of films was there a significant difference in size between expanded and control vessels. Certain other changes were seen and these are illustrated in the angiograms of pig no.7., (Figure 7.5 and 7.6). Very definite arcades of vessels are seen in both flaps however those in the control flap are more tightly packed than in the expanded. Not only are the vessels closer together in the control side but they are considerably more convoluted and as a result there appears to be more vascular marking in the control than in the experimental side which is in contrast to Cherry's findings because at this vessel size there does not appear to be any new vessel formation from skin expansion.

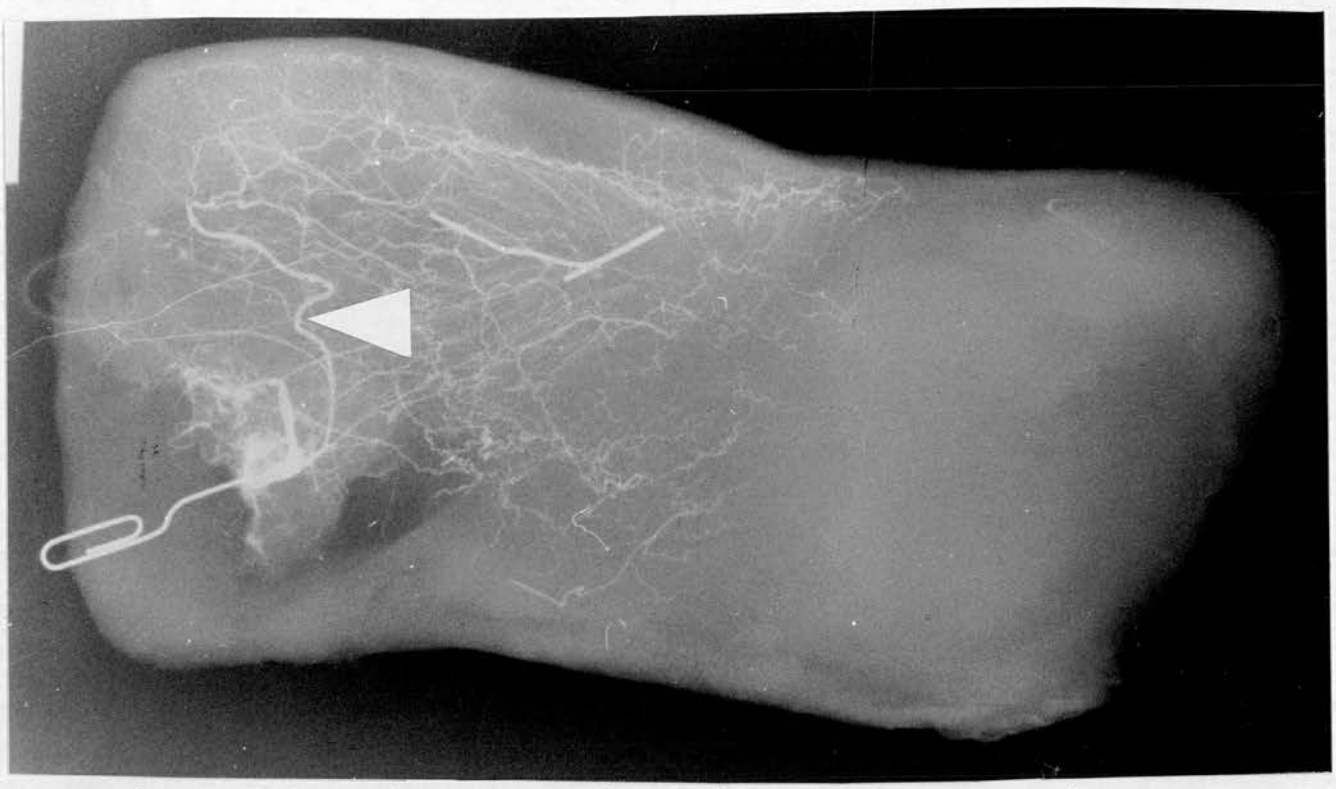


Figure 7.3 Angiogram of Pig No.2 Control Flap, axial

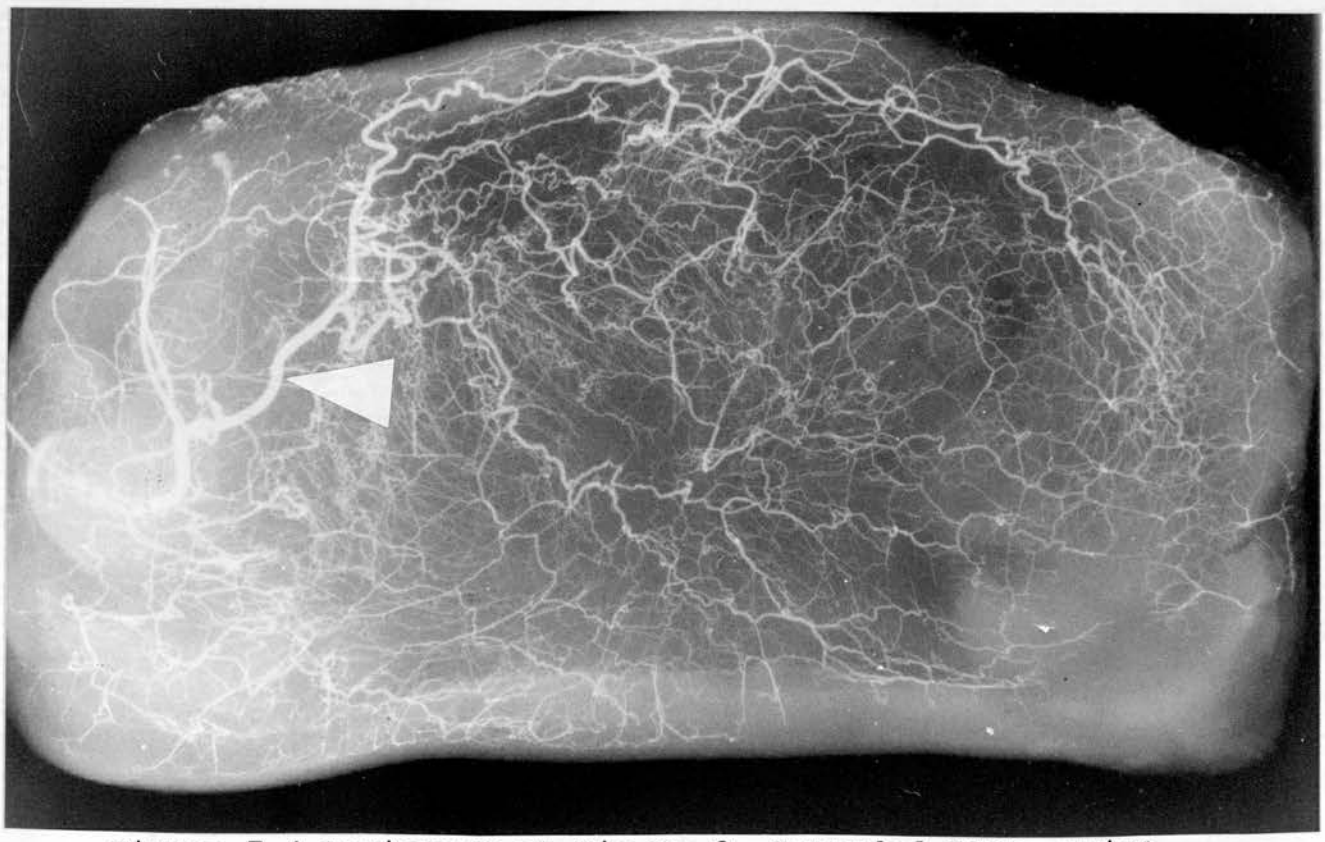


Figure 7.4 Angiogram of Pig No.2. Expanded Flap, axial vessel arrowed.

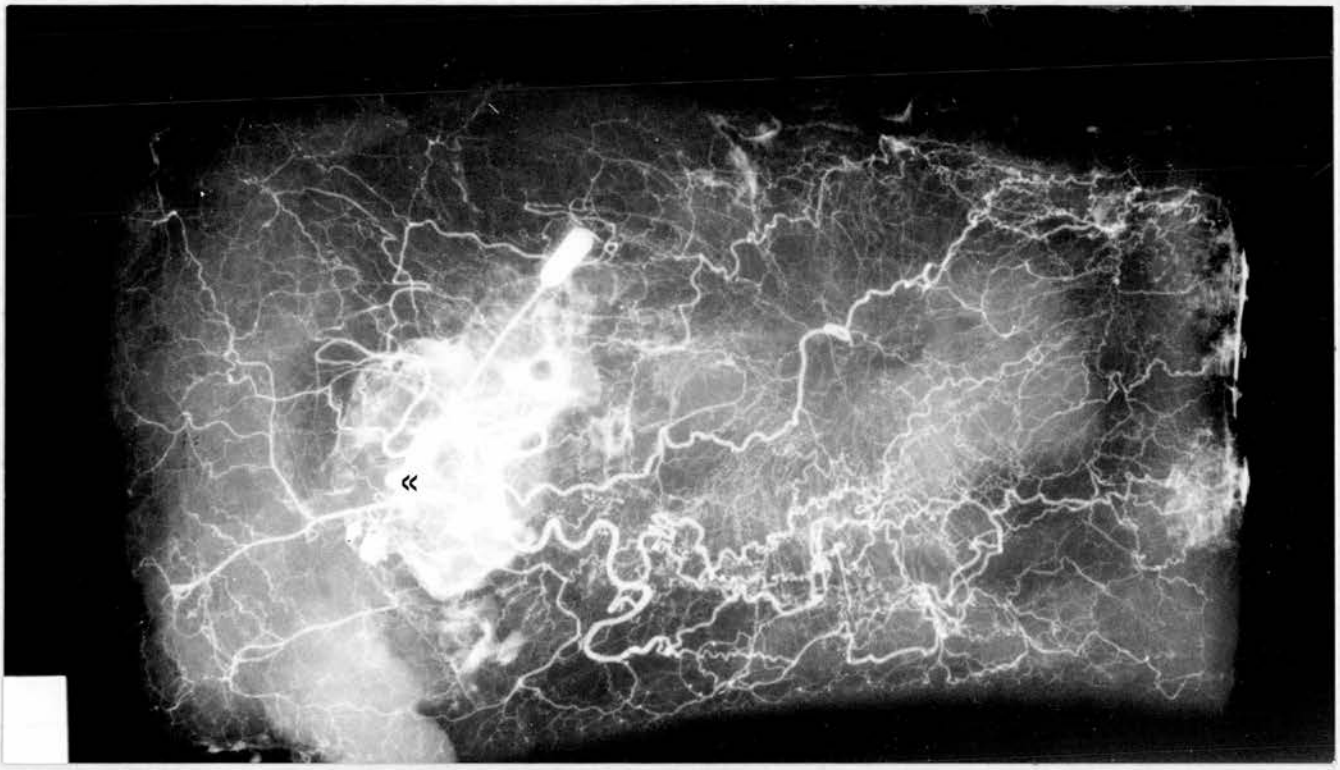


Figure 7.5 Angiogram of Pig No.7, Control Flap, axial vessel arrowed.

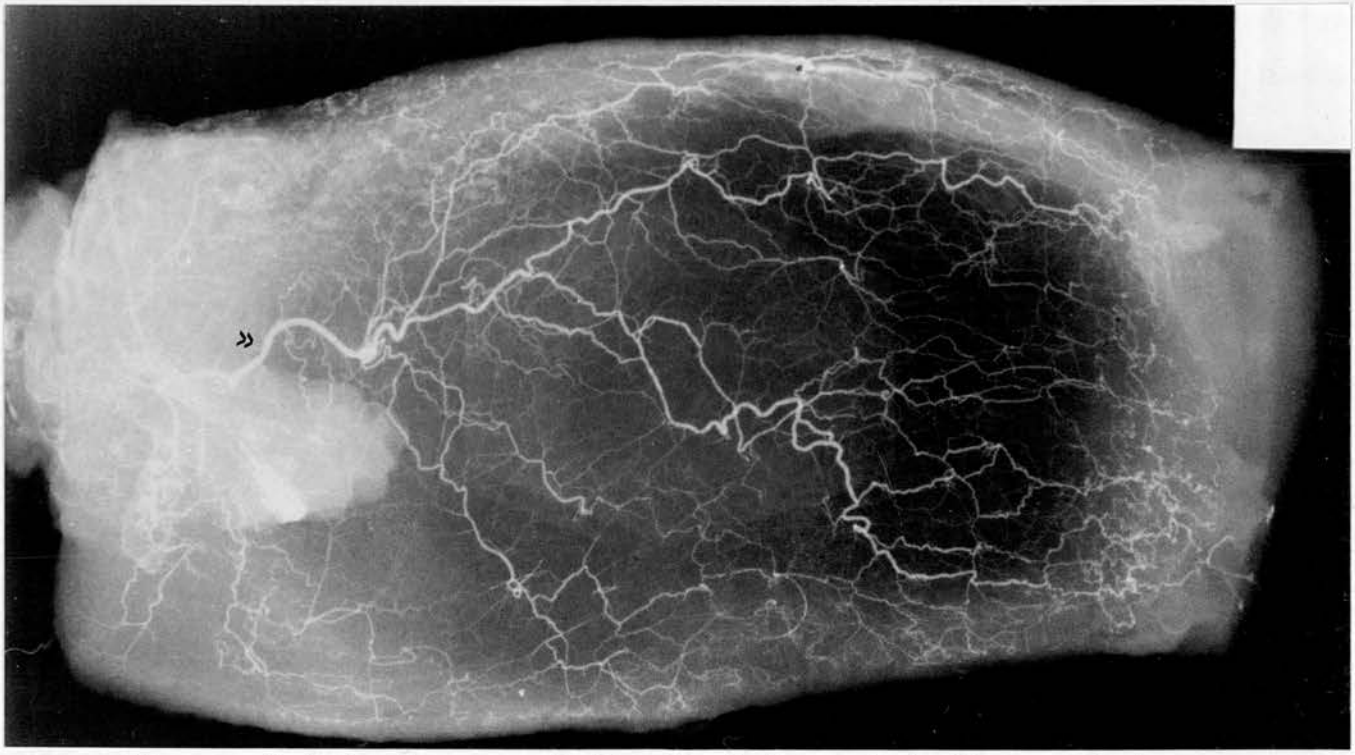


Figure 7.6 Angiogram of Pig No.7 Expanded Flap, axial vessel arrowed.

Distally in the nonexpanded tip of the flap that the vascular changes revert back to normal

Histology

The mean epidermal thickness was found to be increased as a result of tissue expansion, Table 7.1; the mean thickness after expansion being 75 microns and that of the control skin being 62 microns. No difference in the structure or cell types was noted. The irregularity of both the surface of the epidermis and its interface with the dermis was very variable in both expanded and control skin. When multiple measurements were made of the undulation ratio little difference was found between experimental and control sides; mean of the expanded being 1.26 and the control 1.33, (Table 7.2). Sections of epidermis and superficial dermis are shown in Figures 7.7 and 7.8.

Dermal thinning reported after tissue expansion was not seen in this experiment. A change was however seen in the normal three dimensional architecture of collagen whose fibres criss cross at about 70^{o}. In the expanded samples the collagen lattice appeared more elongated as the angles between fibres increased.

The layer of fat was the least resistant to expansion with a reduction of approximately 30% of its thickness, (mean of the expanded 5529 microns and of control 7898 microns, Table 7.1). Sections of flaps of pig no.9 are shown in Figures 7.9 and 7.10 and the whole series graphically in Figure 11.

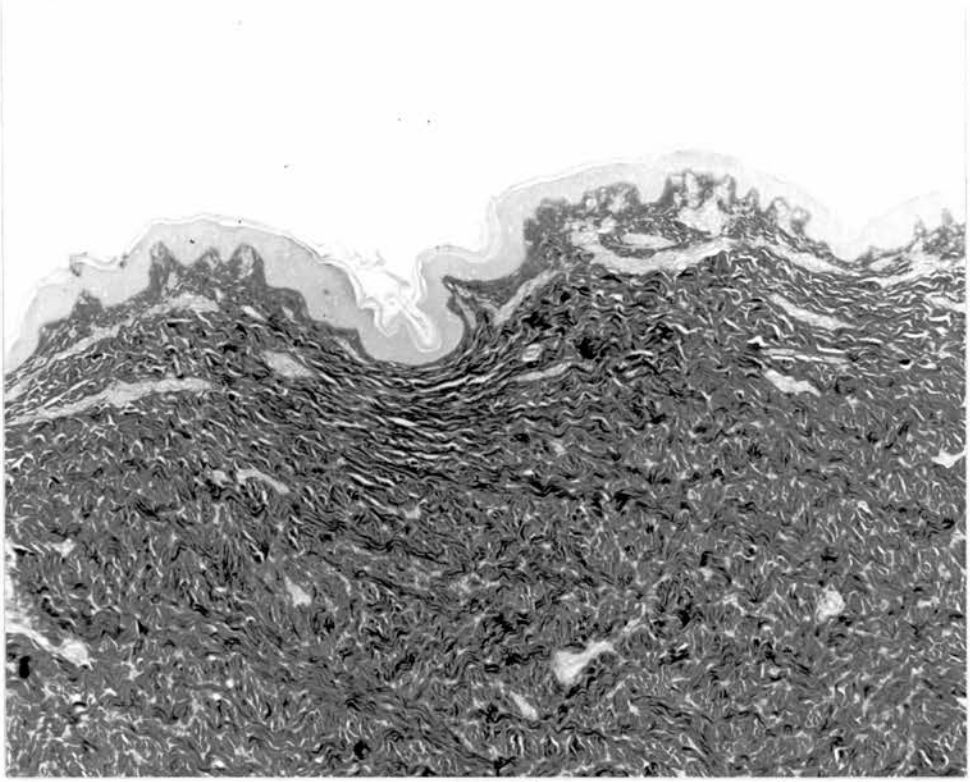


Figure 7.7 Low power H&E sections of epidermis and superficial dermis expanded flap pig no.9

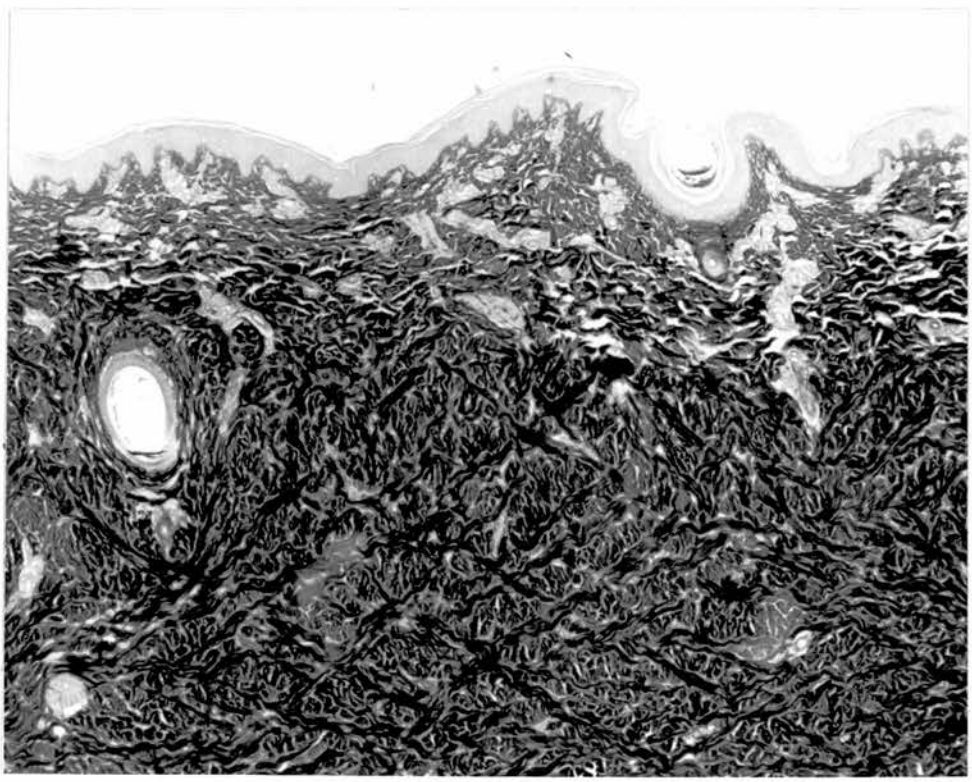


Figure 7.8 Low power H&E sections of epidermis and superficial dermis of control flap pig no.9.

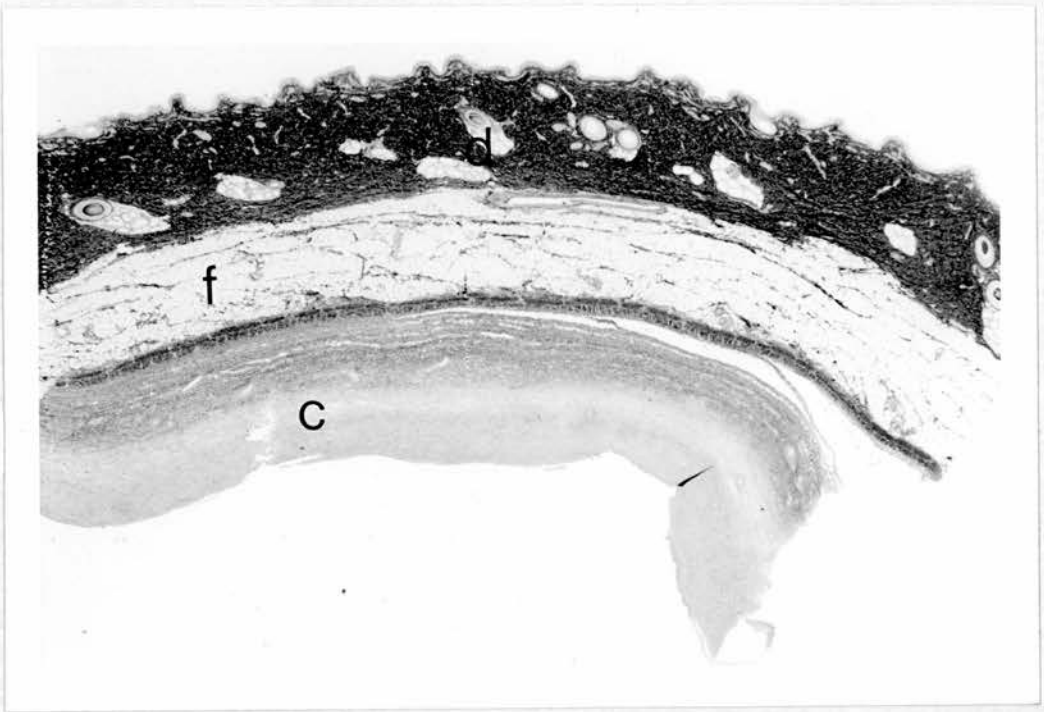


Figure 7.9 Low power sections of expanded flap pig no.9
Dermis - d, Fat - f and Capsule - c.

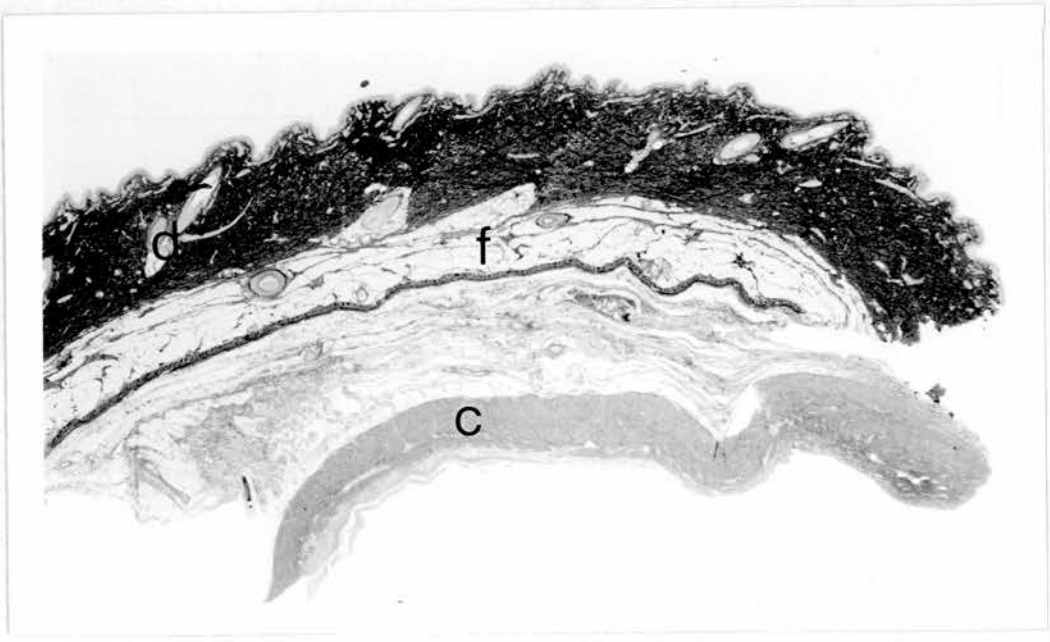


Figure 7.10 Low power sections of control flap pig no.9
Dermis - d, Fat - f and Capsule - c.

TABLE 7.1 **THICKNESS OF SKIN LAYERS** (microns)

	EXPANDED			CONTROL		
Pig	Epid.	Derm.	Fat	Epid.	Derm.	Fat
7	81	2430	4110	63	2892	5843
9	57	2217	3968	63	2192	5305
10	64	2741	7501	82	2685	8844
11	87	2316	5322	45	2120	11697
12	87	2274	6745	56	2183	7802
mean	75	2396	5529	62	2414	7898

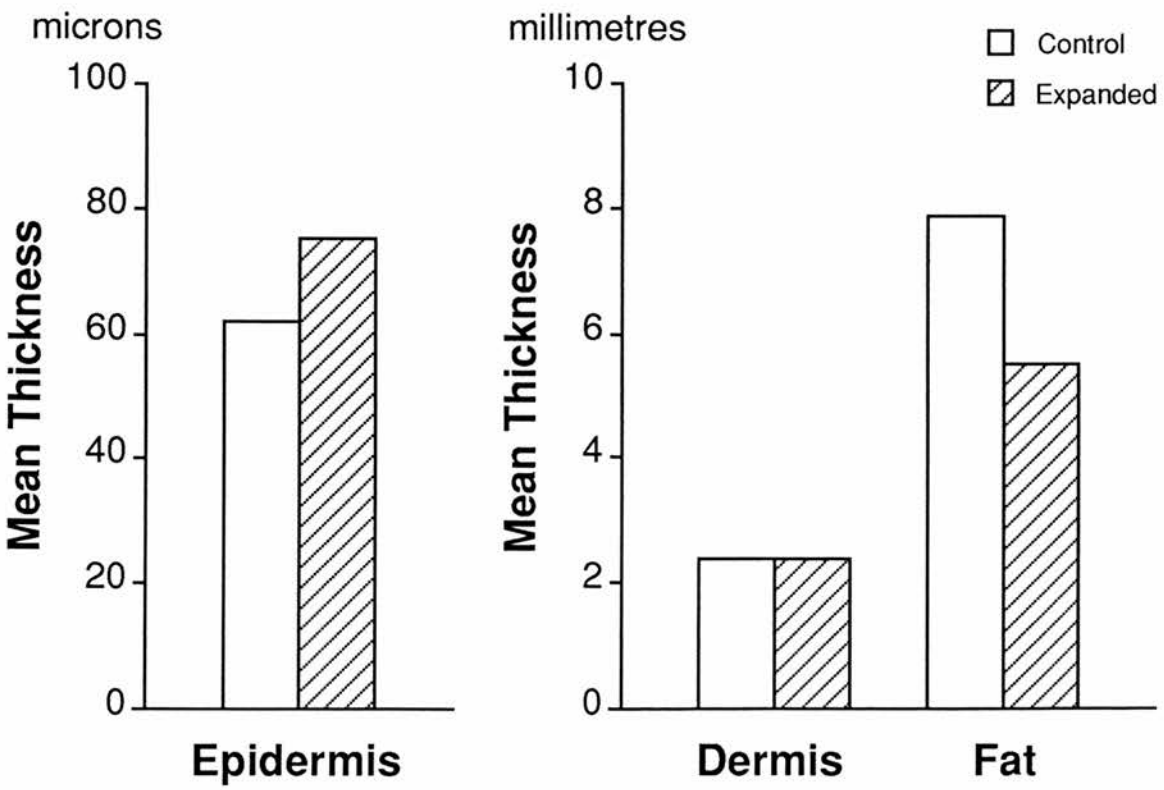


Figure 7.11 Comparison of thickness of components of flap.

TABLE 7.2 **UNDULATION FACTOR**

Pig	Expanded	Control
7	1.34	1.52
9	1.22	1.26
10	1.20	1.31
11	1.22	1.19
12	1.31	1.35
mean	1.26	1.33

A thin layer of panniculus carnosus was present in the middle of the fat. As the distance between this and the capsule was reduced but not the distance between it and the dermis it can be deduced that fat atrophy occurred in the deep surface of the flap.

Despite any difference between the means of expanded and control sides no statistically significant difference was found when any of the above pairs of results were tested.

A capsule rich in collagen fibres was seen in both expanded and control flaps. Few vessels were seen in the capsule itself but by the time inflation had been completed a loose plane had developed between the capsule and the rest of the flap and it was here that vessels were found, (Figure 7.12 and 7.13). This has been denoted the capsular plexus.

Of a vessel size of greater than 500 square microns mean vessel counts revealed an increase in each of the layers studied, (Table 7.3 and Figure 14). However the increase in the number of vessels in the capsular plexus after expansion was the only layer in which the difference was significantly different, ($p < 0.05$).

Similarly the mean luminal area of the vessels was found to be greater in each of the layers in the expanded flaps however this difference was not significant in any, (Table 7.4 and Figure 15).

Figure 7.12 Low power section of Capsular plexus of pig no. 10. Capsule - c and plexus - p. Expanded flap.

Figure 7.13 Low power section of Capsular plexus pig no.10. Capsule - c and plexus - p. Control flap.

TABLE 7.3 **VESSEL COUNTS WITHIN SKIN SAMPLES**

	EXPANDED			CONTROL		
Pig	Derm.	Fat	Caps.Plex.	Derm.	Fat	Caps.Plex.
7	7	7	27	5	7	16
9	15	24	9	14	18	8
10	10	6	25	8	8	15
11	10	16	22	12	10	11
12	17	9	24	9	9	18
mean	11.8	12.4	21.4	9.6	10.4	13.6

TABLE 7.4 **MEAN LUMINAL AREA OF VESSELS WITHIN SAMPLES**

X 10^3 square microns

	EXPANDED			CONTROL		
Pig	Derm.	Fat	Caps.Plex.	Derm.	Fat	Caps.Plex.
7	3.1	2.8	9.3	2.0	7.6	6.1
9	2.6	6.2	8.4	3.2	10.0	3.7
10	1.7	2.1	2.7	1.7	2.6	4.7
11	8.5	3.0	7.5	1.2	7.6	4.9
12	4.5	24.5	14.6	2.1	3.7	4.5
mean	4.1	7.7	8.5	2.0	6.3	4.8

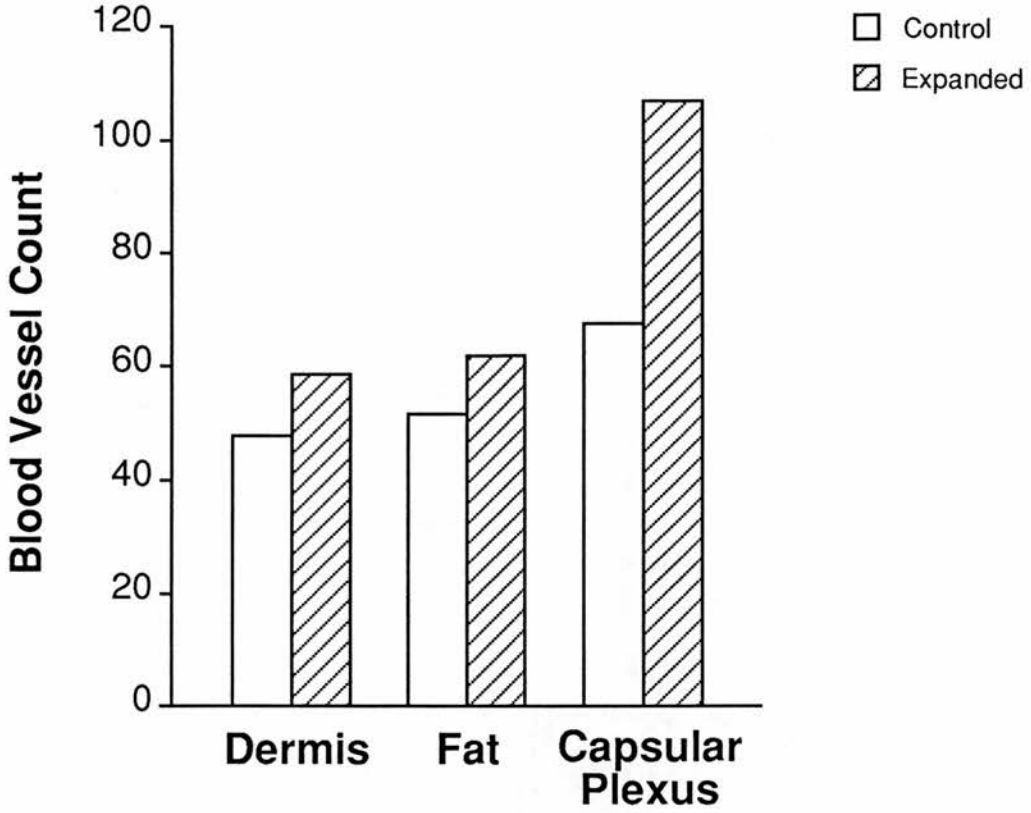


Figure 7.14 Blood vessel counts within flaps

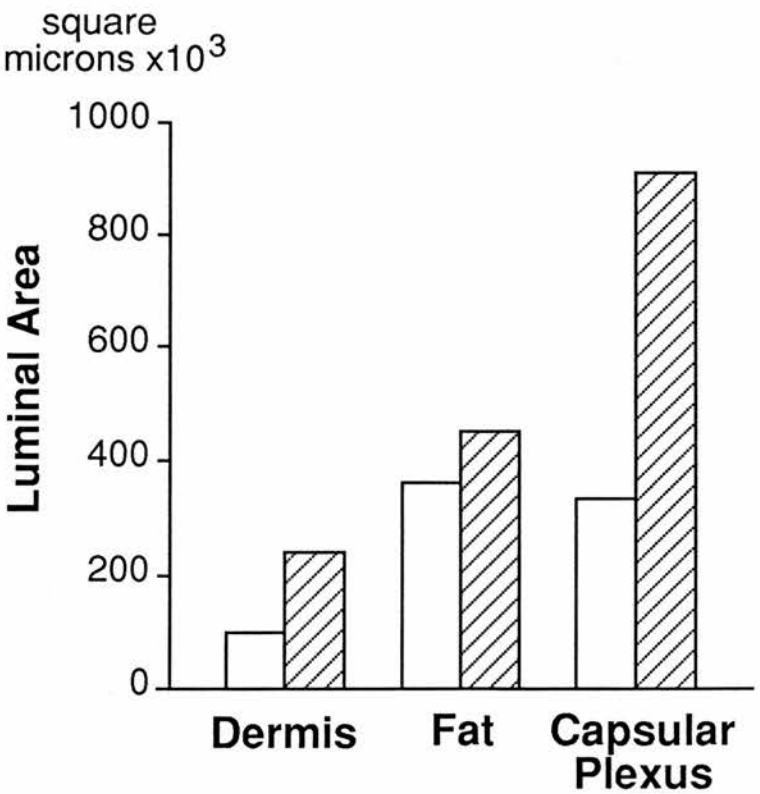


Figure 7.15 Cross sectional area of blood vessel lumen within flaps.

When the vascular pedicles were examined no obvious histological differences were found. As noted by Stark there was no reduction in vessel wall thickness or loss of intimal integrity, (Figure 7.16 and 7.17).

The in vivo studies of axial artery calibre showed the mean diameter of the vessel supplying the expanded flap to be 2.4 mm and that of the control to be 2.38 mm. These figures failed to show any statistically significant difference.

TABLE 7.5 MEASUREMENT OF AXIAL ARTERY IN VIVO

Diametre in mms.

Pig	Expanded	Control
7	2.15	2.10
9	2.20	2.05
10	2.90	3.05
11	2.50	2.50
12	2.25	2.20
mean	2.40	2.38
8*	3.20	3.00

(* infected)

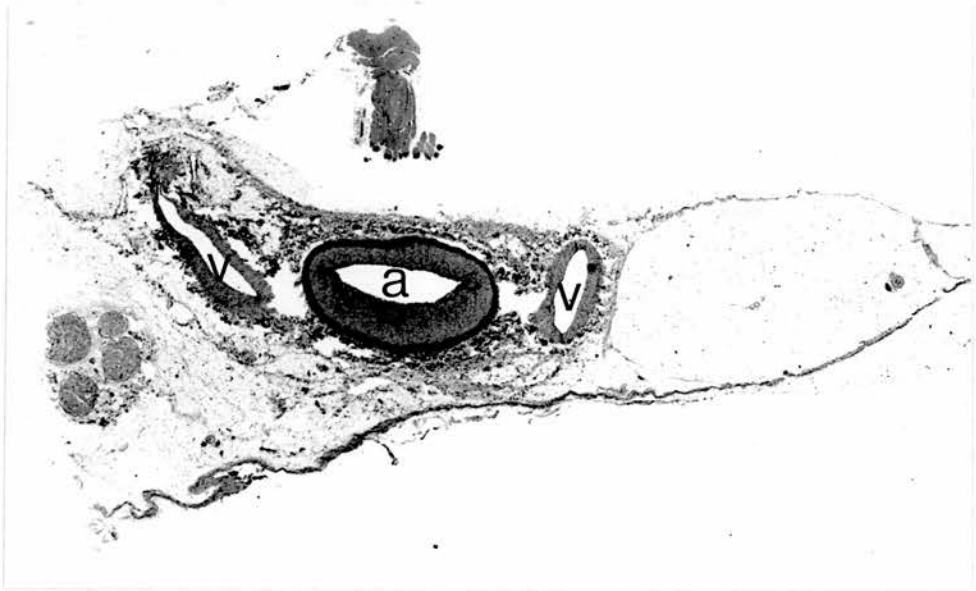


Figure 7.16 Low power section of vascular pedicle pig no.9. Artery - a and Veins - v. Expanded Flap.

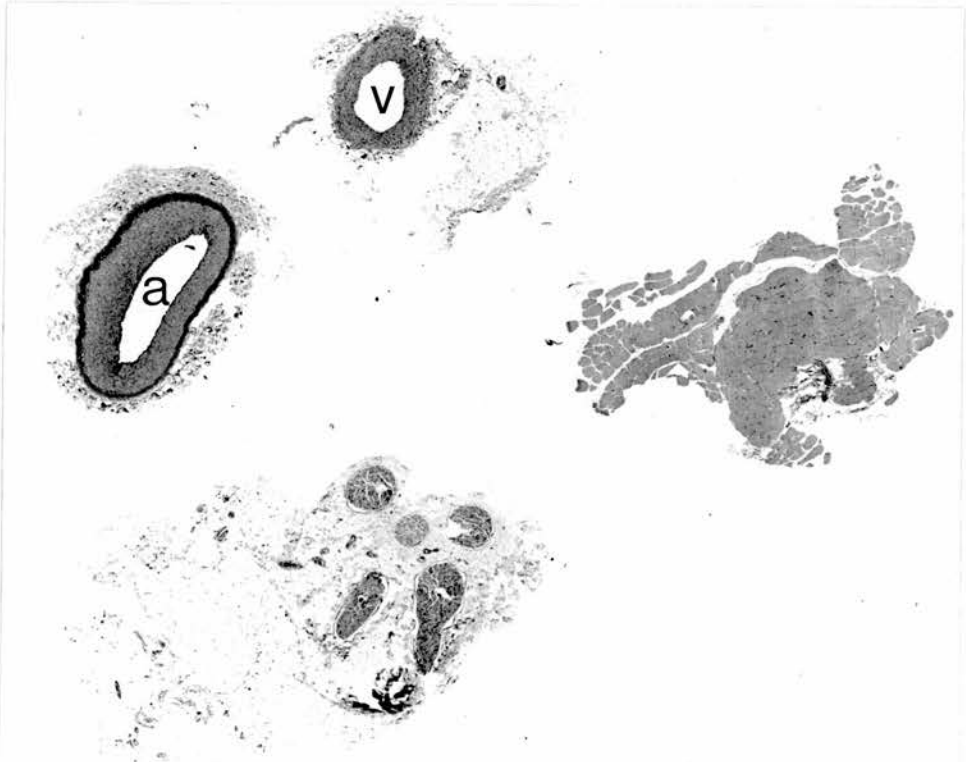


Figure 7.17 Low power section of vascular pedicle pig no.9. Artery - a and Veins - v. Control flap.

DISCUSSION

It was not appreciated until the second pair of angiograms were examined that the appearance of the vascular tree as seen on the radiograph could be dramatically affected by the volume of dye, the speed at which injected and the delay between injection and X ray exposure. If the first two factors were increased or the last decreased the calibre of vessels seen on the radiographs would appear greater and there would be much better penetration of dye into the small vessels of the vascular tree. Therefore dye injection was standardised for the last three pairs.

Image analysis by tracing outlines on a digitising tablet linked to a computer has become a recognised method of measuring lengths and areas of complicated geometrical shapes. It has been developed primarily for use in cartography. By using the pen to trace across the epidermis, dermis and subcutaneous tissues it was simple to determine thickness values. As the depth and density of the rete pegs varied quite considerably within one single 2 cm. skin sample it was necessary to take many measurements of epidermal thickness to achieve a meaningful result. In the absence of tissue generation stretching of skin would presumably spread the rete pegs apart and the undulation of the interface between epidermis and dermis would be reduced, corresponding to the changes found by Van Rappard, (1988). This was sought

by measuring the "undulation factor" in addition to measuring tissue thickness.

The measurement of external diameter of the axial vessels was made in the first pig by an image splitting device. This is an optical instrument which can double the image of a linear structure such as a hair or a small blood vessel. The two images are then separated and the distance they need to be separated so that they lie immediately adjacent to each other is equal to the diameter of the structure. This equipment was cumbersome to use over the anaesthetised pig and its accuracy was lost by the movement of the pig during respiration. It was found to be simpler and more easily reproducable to use Vernier calipers.

7.5 SUMMARY

Under control conditions angiography of the flaps' vascular tree demonstrated that the vessels reached the distal part of the expanded flaps by a process of elongation. Although this may have been achieved partly by growth it was seen that a major contributory factor was the unravelling of the normally tortuous cutaneous vessels.

Histology revealed that some of the changes previously reported had occurred. The epidermis became thicker and the fat layer thinned although neither of these were statistically significant. The rate of undulation at the interface of dermis and epidermis was unchanged though this was seen to be so variable along even a short section of skin that this cannot be used as evidence that the epidermis maintains its structure by growth of new rete pegs and thus new tissue.

The dermis was not seen to thin however a reorientation of the collagen fibres within was apparent. A typical fibrous but not particularly vascular capsule was seen but there was a definite vascular plexus noted between the capsule and the overlying flap tissues. This has been denoted the capsular plexus.

In the microscope sections an increase in the number of vessels greater than 500 microns^2 was seen in all layers of the flap but only achieved statistical significance in the capsular plexus. Similarly there was

an increase in the mean size of the vessels in all layers although again this did not reach statistical significance.

No change was found in the microscopic structure of the vessels studied histologically or in their diameter measured in vivo following tissue expansion.

CHAPTER 8 DISCUSSION

8.1 EXPERIMENTAL CONCLUSIONS

8.2 CLINICAL APPLICATION

8.3 FUTURE DEVELOPMENTS

8.1 Experimental Conclusions.

The first part of this study conclusively shows that as a direct result of tissue expansion the territory of a specific cutaneous blood vessel is enlarged in terms of surface area. Survival of the expanded island flaps was similar to that of the expanded random pattern flaps of Cherry. In practice therefore a larger island flap can be raised without concern that any increase in flap area may depend on random vessels entering the flap at its periphery.

The second and third parts of this study were aimed at determining how the increased area of skin could survive. Previous studies have claimed that tissue expansion causes an increase in the number of small cutaneous vessels, (Cherry 1983), an increase in blood flow, (Leighton 1988), and an increase in the calibre of the feeding arteries, (Min 1988).

In chapter 6 the blood flow through the deep circumflex iliac artery was measured and no difference was found when that through the expanded flaps was compared to the control flaps. This is in direct contrast with the findings of another study which was published during the course of this project, (Leighton et al 1988). In Leighton's work expanders were inserted under pairs if pig buttock flaps in a very similar experiment. Flow

through the deep circumflex iliac artery was then measured, again using electromagnetic flowmetry and the two sides compared. Average flow rates of 4.5 ml./min. were found in their control flaps but the expanded flaps had a significantly higher flow at 8.1ml./min.. It was surprising that the flow rates in general were considerably lower than in the present project, especially considering that the size of the pigs was similar. There was however one major difference in experimental protocol which may account for the considerable variation in Leighton's results. In the present study blood flow measurements were made with the flaps left undisturbed in situ and with only minimal dissection of the axial vessels whereas in Leighton's study the flaps were raised and islanded on their vessels before flow rates were measured.

Although it is known that blood flow through an axial vessel may be increased when the flap is islanded, (McKee et al, 1982), once islanded a flap will lose heat fast and consequent vasoconstriction will soon cause a net reduction in blood flow. Most important of all however was found to be the effect of tension on the vascular pedicle itself. In the present study it was found that even without raising the flap any slight kinking of or traction on the vessel, as might be caused by the weight of the probe, would cause a massive reduction or even cessation of blood flow. It was obvious that had the flap been islanded and suspended, as in

Leighton's study, it would be extremely difficult to make measurements under identical conditions of evaporative heat loss and tension within the axial vessels.

In parallel to the failure to find any increase in blood flow in expanded skin flaps no increase in temperature was found in the skin of these flaps which might have been expected to accompany hyperaemia.

When the arterial tree of the flaps were mapped out with angiograms the need to standardise injection techniques became evident. Under strictly controlled conditions the changes of neovascularisation seen by Cherry were not observed. In contrast the vessels appeared to be less dense as the skin was expanded. The angiography demonstrated a stretching out of the vascular tree rather than an increase in the length of the larger cutaneous vessels. Measurement of flap weight was used as a simple although indirect measurement of tissue bulk and the increased weight of the expanded flaps would suggest that the elongated vascular tree is distributed within an increased bulk of tissue which, in the absence of significant tissue oedema or recruitment of adjacent skin, must be due to the generation of at least some new tissue.

In the histological specimens proliferation of smaller vessels of a diameter of 20 - 200 microns was observed. Not only was there an increase in the number of small vessels throughout the dermis and fat but a definite plexus of vessels was apparent between the

capsule and adjacent fat. As this vascular plexus is arranged in a horizontal plane it would assist in perfusing the periphery of the flap.

No increase in calibre of the feeding vessels could be demonstrated in vivo or histologically in contrast to the work of Min, (1988). However again there were differences in experimental protocol which may account for quite wide variations in her particular study. An identical experimental model was used but the calibre of the axial vessel was studied using in vivo angiograms made by retrograde catheterisation of the femoral artery at the time of expander insertion. For technical reasons the femoral artery had to be ligated at this point and this stimulated a great network of collateral vessels to open up. As the deep circumflex iliac artery was one of the principle ones it is not surprising that considerable increase in calibre was found when vessel was measured again at the completion of expansion. Great doubt must therefore be cast on comparisons between expanded and control sides in this study.

As blood flow through skin is many times greater than the minimum required for its survival, (Keele et al 1982), an increase in blood flow may not be the critical factor which enables the increased area of skin survival seen after tissue expansion. The mechanism by which the distal parts of the expanded flap survive may be better explained by a change in the architecture of the arteries. As the vessels become less tortuous they become

more axial in nature and thus more effective at delivering blood over greater distances, although there may be an element of true vessel elongation, (Stark 1988).

In summary there appears to be an increase in the size of the vascular compartment of expanded skin restricted to the small vessels only. In the absence of an increased blood flow entering the skin this suggests that the blood flow within the skin is not in fact hyperaemic and that the erythema sometimes seen in expanding skin can be explained by the fact that there is sluggish flow within an enlarged vascular compartment. This effect may therefore not be the beneficial physiological phenomenon as originally claimed by Radovan.

8.2. Clinical Application

The design of flaps in expanded skin is more complicated than elsewhere due to the extreme convexity of the surface of the expander. In order to make maximum use of expanded skin with the minimum of scarring it is usually necessary to raise a definite flap from one side of the expander rather than trying to utilise the expanded skin by simple advancement. This involves a clear appreciation of the complicated geometry involved. Little guidance has been given on rules governing the design of complicated skin flaps. It is seen that the effect of delay produced by expander insertion will enable a longer flap to be raised than that possible acutely. The increase permissible being similar to that achieved by conventional bipedicled delay.

Following tissue expansion a further increase in surviving flap length is possible and this is exactly proportional to the total increase of area produced by the expansion. Thus if the design of a flap was to be tattooed into skin previously delayed by the insertion of an an expander and if viable before expansion it would survive following expansion regardless of where or how much the skin has been expanded. However the surgeon should not expect any increased survival beyond these limits and he should in addition bear in mind that there may be some reduction in perfusion per unit weight of tissue.

When a flap in expanded skin has been designed debate still continues over whether the capsule should be

included in the flap or not. Capsulotomy or even capsulectomy has been advocated as a means of "persuading" expanded skin flaps to reach further, (Leonard and Small 1986), but others contend that the capsule is highly vascular and its removal will reduce the flap's blood supply, (Manders 1984). In this study the capsule itself was not found to be highly vascular but great care should be exercised if it is to be removed in order to avoid damaging the vascular plexus found immediately adjacent.

The use of pedicled island flaps in plastic surgery is relatively limited however as all free tissue transfer involves flaps which can be islanded conclusions from this study can be applied to free flaps also. Although no microvascular anastomoses were performed in this study there is no reason to suspect that the structure of the vascular pedicle at several centimetres proximal to the site of expansion is affected in any way that might be detrimental to a vascular anastomosis. It has even been found that expansion of the vessel at the site of the anastomosis itself is possible, (Stark 1988).

The possibility is therefore raised of expanding flaps intended for free tissue transfer prior to transfer. As the size of an island or free flap that can be raised in clinical practice is, in part, limited by donor-site morbidity there are obvious attractions. There would be the possibility of a larger free flap for reconstruction and, if some expanded tissue is left

behind at flap transfer, a donor site which might have required a skin graft could possibly be closed directly. Although the initial impression that the axial vessel would dilate during tissue expansion enabling an easier and safer anastomosis, (Saxby 1988), this does not unfortunately appear to be the case. However the capsule of the expanded flap may be a benefit as once this is identified and entered the flap may be swiftly and easily raised at the time of transfer. The capsule provides a supportive and well vascularised backing to the flap. Thus examples of pre-expanded free tissue transfer have already been reported,(Leighton et al 1988), and the following case report is presented.

Case Report 6

A 17 year old male was fell off a motor cycle injuring his left heel. He lost an area of skin 14 cms. by 12 cms. over his tendo achilles and os calcis, (Figure 8.1). This was treated primarily by a split skin graft but 12 months later he continued to have problems with an unstable scar which was tethered to the underlying tendon, (Figure 8.2). A plan was made to resurface the area with an expanded forearm flap to be transferred to his leg by a microvascular technique. At a preliminary operation a 500ml. round expander was placed under the fascia of his right forearm under an area of skin that can be raised as a free flap based on the radial artery, (Song 1979). This flap was then expanded to a volume of 800mls. before it was raised on its vessels,(Figure 8.3).

After removal of the skin graft from the heel and preparation of the posterior tibial vessels a large flap 10 cm. by 12 cm. was transferred to the left ankle. The large amount of forearm skin generated permitted direct closure of the forearm donor site, Fig.8.4. The heel is shown 4 months later with good durable skin cover and the forearm is also soundly healed, Fig.8.5.

Figure 8.1 (Left) Heel one week after injury

Figure 8.2 (Right)Heel with unstable skin graft.

Figure 8.3 Expansion of Radial Forearm Flap

Figure 8.4 Primary closure of donor site.

Figure 8.5 Final reconstruction of left heel.

The clinical indications for pre-expanding a free flap may not often occur.It is unlikely that many defects which are serious enough to warrant free tissue transfer will be able to wait the two months necessary for tissue expansion with perhaps the exception of burns scarring. It has been questioned whether our ability to guarantee success in both tissue expansion and microvascular surgery have reached such a level that after investing two months and an initial operation we can justify risking the whole effort in free tissue transfer,(Van Beek and Adson 1988). An alternative solution to some problems would be the conventional transfer of a free flap and subsequent of skin locally around the donor site

to remove any temporary skin graft that might have been necessary. There are two occasions when this might not be possible and pre-expansion of a free flap would be indicated. Firstly when a flap larger than that which can safely be raised as a conventional free flap is required. Secondly if the free flap required is so large that its transfer would remove so much skin locally that tissue expansion of the remainder would be very difficult. This situation might arise if a very large radial flap was raised leaving only a small strip of skin on the forearm.

8.3 FUTURE DEVELOPMENTS

This project raises perhaps the most fundamental question still to be answered in Tissue Expansion - How much of the increased tissue area is due to generation and how much is due to stretching of existing tissues? Probably some element of both occurs and the relative proportion of each may depend on the rate at which expanded, the period for which left fully expanded and, in particular, individual skin characteristics. As any obstetrician knows the amount of redundant abdominal skin remaining following pregnancy is very variable and not totally related to the size of the pregnancy; in some women there is scarcely any skin laxity and in others there more than enough to warrant abdominoplasty.

It is interesting to note that the percentage of flap survival remains constant whether tissue expansion has taken place or not, contrary to that found by others, (McCann 1988). If only tissue stretching has occurred with no new tissue generation this might be expected because the vessels would presumably supply exactly the same tissue following expansion only redistributed in a stretched and thinner form. However if new tissue is generated somehow the vascular tree will not only enlarge directly proportional to the degree of expansion but become able to sustain the extra tissue produced.

If the answer to this question is found it may be possible to direct skin expansion towards tissue

generation rather than tissue stretching which in turn should reduce the late contraction of expanded skin the detrimental effect of which is often seen. It is possible that this might be achieved by altering physical variables such as the rate and duration of expansion or by pharmacological manipulation. Increased skin expansion has already been shown following instillation of anticontractile agents into the expander cavity, (Lee et al 1985 and Joseph et al 1988), perhaps other growth promoting factors could be harnessed to produce a beneficial effect.

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APPENDICES

APPENDIX 1

EXPANSION RATES OF PIGS

Pig	Day	Preinflation Pressure mm.Hg.	Cumulative Volume ml.	Postinflation Pressure mm. Hg.
1	0	---	100	---
	7	7	200	21
	14	6	280	25
	21	5	380	20
	25	8	480	27
	29	5	570	20
	36	5	670	28
	43	8	750	27
2	0	---	200	---
	7	---	370	---
	14	4	460	19
	21	4	610	29
	28	3	740	21
	33	5	740 (tube ? kinked)	
	38	3	850	28
	48	8	950	32
	56	5	1000	8

Pig	Day	Preinflation Pressure mm.Hg.	Cumulative Volume ml.	Postinflation Pressure mm.Hg.
3	0	---	200	---
	7	---	300	---
	14	---	410	---
	21	9	480	27
	28	---	560	---
	35	---	620	---
	42	---	690	---
	46	---	770	---
	49	---	840	---
	56	---	910	---
	61	---	1000	---
4	0	---	150	---
	7	3	290	21
	14	8	370	17
	17	7	410	19
	21	6	470	14
	35	4	590	19
	39	---	730	---
	45	---	830	---
	52	7	910	17
	59	---	1000	---

Pig	Day	Preinflation Pressure mm.Hg.	Cumulative Volume ml.		Postinflation Pressure mm.Hg.
5	0	---	200		---
	7	---	300		---
	14	---	450		---
	21	---	670		---
	28	---	900		---
	35	---	1000		---
6	0	---	150		---
	7	15	210		19
	14	15	320		22
	17	---	380		---
	21	---	380	?tube kinked	---
	28	---	350	?tube kinked	---
	33	---	440		---
	38	---	600		---
	42	---	720		---
	49	---	850		---
	56	---	1000		---

Pig	Day	Preinflation Pressure mm.Hg.	Cumulative Volume ml.	Postinflation Pressure mm.Hg.
7	0	---	200	---
	7	11	380	26
	14	13	480	---
	21	---	580	---
	28	---	710	---
	35	---	830	---
	42	9	1000	13
8	0	---	200	---
	7	7	400	21
	14	---	500	---
	17	---	650	---
	21	---	800	---
	35	---	900	---
	42	---	1000	---

Pig	Day	Preinflation Pressure mm.Hg.	Cumulative Volume ml.	Postinflation Pressure mm.Hg.
9	0	---	100	---
	10	8	160	16
	14	---	250	---
	19	---	350	---
	21	---	410	---
	25	6	480	14
	28	6	600	20
	35	---	740	---
	42	---	830	---
	49	---	930	---
	56	---	1000	---
10	0	---	100	---
	7	---	280	---
	14	---	390	---
	21	14	500	24
	28	---	620	---
	33	---	720	---
	40	10	820	---
	45	---	920	---
	56	---	1000	---

Pig	Day	Preinflation Pressure mm.Hg.	Cumulative Volume ml.	Postinflation Pressure mm.Hg.
11	0	---	100	---
	7	---	220	---
	14	---	320	---
	19	---	440	---
	24	---	500	---
	28	---	560	---
	35	---	700	---
	42	---	820	---
	49	---	910	---
	56	---	1000	---
12	0	---	150	---
	7	9	250	17
	14	---	350	---
	21	---	450	---
	28	---	570	---
	35	11	690	21
	42	---	750	---
	49	---	850	---
	56	---	930	---
	64	---	1000	---

APPENDIX 2

HISTOLOGICAL MEASUREMENTS SERIES 2

SKIN LAYER THICKNESSES

EPIDERMIS, (microns),

Pig 7:

Expanded: 69, 82, 118, 55, 129, 81, 77, 34, 99, 275, 98, 90.

Control : 52, 45, 91, 46, 47, 67, 61, 63, 61, 101, 39, 84.

Pig 9:

Expanded: 62, 74, 62, 43, 63, 43, 40, 47, 86, 66, 53, 45.

Control : 111, 55, 29, 86, 50, 53, 43, 75, 45, 102, 39, 72.

Pig 10:

Expanded: 93, 70, 108, 43, 46, 39, 59, 55, 64, 66, 56, 67.

Control : 50, 179, 79, 76, 64, 75, 60, 74, 105, 54, 75, 90.

Pig 11:

Expanded: 68, 162, 60, 99, 71, 71, 89, 43, 103, 76, 122,101.

Control : 52, 44, 40, 54, 18, 42, 36, 71, 56, 35, 54, 36.

Pig 12:

Expanded: 100, 88, 79, 83, 63, 62, 86, 54, 159, 95, 76, 98.

Control : 56, 35, 50, 80, 93, 40, 39, 54, 45, 54, 75, 49.

FAT, microns,

Pig 7:

Expanded: 3407, 4895, 4132, 4006.

Control : 6481, 4920, 4378, 7593.

Pig 9:

Expanded: 4103, 3658, 3802, 4309.

Control : 6462, 4445, 5604, 4707.

Pig 10:

Expanded: 7611, 7280, 7144, 7969.

Control : 8990, 9425, 7943, 9018.

Pig 11:

Expanded: 5296, 5408, 4323, 6261.

Control : 11495, 10469, 12914, 11910.

Pig 12:

Expanded: 7218, 6800, 6975, 5987.

Control : 7026, 7181, 8711, 8290.

DERMIS, (microns),

Pig 7:

Expanded: 2907, 2341, 1938, 2548.

Control : 3225, 2974, 2519, 2581.

Pig 9:

Expanded: 2031, 2361, 2171, 2255.

Control : 2075, 1987, 2293, 2412.

Pig 10:

Expanded: 2764, 2886, 2105, 3210.

Control : 2637, 2896, 2497, 2710.

Pig 11:

Expanded: 2247, 2131, 2574, 2310.

Control : 2288, 2227, 1783, 2181.

Pig 12:

Expanded: 2236, 2029, 2388, 2441.

Control : 2560, 2413, 1913, 1847.

UNDULATION RATIO,

Pig 7:

Expanded: 1.32, 1.45, 1.34, 1.24.

Control : 1.20, 1.69, 1.56, 1.61.

Pig 9:

Expanded: 1.29, 1.27, 1.18, 1.15

Control : 1.30, 1.17, 1.28, 1.28.

Pig 10:

Expanded: 1.28, 1.05, 1.19, 1.26.

Control : 1.42, 1.29. 1.30, 1.21.

Pig 11:

Expanded: 1.39, 1.18, 1.08, 1.24.

Control : 1.20, 1.31, 1.15, 1.08.

Pig 12:

Expanded: 1.26, 1.20, 1.46, 1.36.

Control : 1.40, 1.27, 1.39. 1.35.

CROSSECTIONAL AREA OF VESSELS OF SERIES 2 (square microns)

Pig 7, Expanded,

Dermis: 2137, 1362, 2445, 701, 4214, 3558, 7304

Fat: 1661, 3669, 2273, 1948, 6666, 2809, 868.

Capsular plexus: 1291, 5357, 1856, 2228, 22707, 2075, 4721, 13676, 35022, 1918, 5066, 19697, 11555, 931, 16090, 35724, 20137, 11008, 3020, 2462, 1468, 2290, 1534, 4092, 3516, 6368, 14103.

Control,

Dermis: 1367, 1005, 848, 5250, 1645.

Fat: 12993, 4026, 938, 2528, 3762, 1027, 27674.

Capsular Plexus: 3705, 3119, 2622, 3762, 867, 1331, 9218, 24387, 709, 942, 1499, 5855, 29638, 5120, 770 3439.

Pig 9, Expanded

Dermis: 867, 1002, 9306, 3041, 1413, 864, 2865, 2568, 1861, 1169, 6717, 590, 1102, 3144, 1921.

Fat: 1738, 798, 1290, 719, 2105, 1628, 15290, 2098, 7686, 867, 1120, 22113, 921, 1350, 1413, 9621, 994, 6108, 4381, 8767, 767, 4120, 6555, 46546.

Capsular Plexus: 22120, 968, 995, 3467, 4481, 760, 19000, 4098, 19873.

Control

Dermis: 2561, 8862, 1691, 642, 2191, 6221, 1433, 1296, 5567, 1492, 1283, 3356, 897, 6622.

Fat: 8377, 2336, 2426, 2012, 2231, 11267, 9716, 3175, 14385, 4939, 16169, 7272, 1713, 6312, 13001, 5183, 4340, 64498.

Capsular Plexus: 1119, 689, 3549, 1493, 3602, 8894, 4680, 5638.

Pig 10, Expanded

Dermis: 1250, 1405, 2506, 1193, 1070, 737, 1070, 756, 809, 5734.

Fat: 1434, 2095, 1463, 1729, 2333, 3588.

Capsular Plexus: 1925, 1123, 1918, 2715, 1904, 1667, 1958, 4146, 3020, 6500, 614, 1414, 6581, 6348, 928, 1829, 2453, 2395, 870, 1274, 725, 3445, 5029, 785, 6284.

Control

Dermis: 1324, 1627, 1803, 796, 605, 1462, 4361, 1710.

Fat: 1656, 941, 643, 576, 689, 11460, 1161, 3762,

Capsular Plexus: 1568, 1064, 23428, 1087, 5130, 15339, 9110, 5682, 868, 1490, 2410, 1010, 981, 1063, 765.

Pig 11 Expanded

Dermis: 1243, 2746, 2664, 5631, 2021, 2157, 56215, 5323, 1189, 6031.

Fat: 1816, 980, 763, 1967, 718, 2612, 4190, 2186, 3985, 1544, 766, 1039, 3425, 1273, 1932, 19588.

Capsular Plexus: 3657, 1528, 1262, 1266, 1425, 1139, 12170, 1523, 1970, 1571, 2156, 28697, 1768, 2584, 2284, 608, 75003.

Control

Dermis: 1706, 808, 1454, 532, 634, 734, 594, 919, 1053, 1533, 1843, 2362.

Fat: 18604, 1466, 10110, 986, 34718, 952, 1914, 544, 3620, 2616.

Capsular Plexus: 4420, 2307, 1348, 6740, 2216, 897, 681, 1101, 1416, 934, 31820.

Pig 12, Expanded

Dermis: 795, 693, 591, 5464, 522, 5974, 512, 3201, 639, 16431, 1956, 3024, 5530, 14443, 2029, 1703, 12772.

Fat: 1481, 1487, 7525, 3407, 5510, 151124, 5548, 20183, 23947.

Capsular Plexus: 2734, 24605, 5523, 3501, 29938, 20610, 3518, 3911, 989, 2702, 1712, 58570, 20656, 15984, 2011, 44022, 16067, 5411, 2563, 1223, 2511, 10499, 69423, 1789.

Control

Dermis: 889, 1133, 4743, 1763, 3824, 626, 2838, 645, 2547.

Fat: 1159, 5133, 3306, 10876, 1212, 3031, 6154, 1954, 547.

Capsular Plexus: 1395, 800, 659, 1057, 1738, 5782, 4041, 2397, 605, 526, 1416, 2234, 2500, 3814, 1300, 3992, 626, 46393.

APPENDIX 3

Survival of Island Flaps after Tissue Expansion: A Pig Model

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Survival of island flaps after tissue expansion has been studied. Expanders were placed under each buttock flap of six minipigs and one side was expanded while the other was left empty as a control. Both flaps were then raised and isolated on their vascular pedicles in order to compare flap survival 7 days later. It was found that the survival lengths of the expanded flaps were approximately 50 percent greater than those of the delayed controls. Microangiography suggested that the diameter of the axial artery increased following expansion.

In clinical practice this technique would provide a larger flap for reconstruction and the possibility of direct closure of the donor site. In addition, the observed increase in vessel caliber should facilitate the free tissue transfer of expanded flaps.

The size of an island or free flap that can be raised in clinical practice is, in part, limited by donor-site morbidity. Controlled tissue expansion is a method of increasing flap size and providing the possibility of direct closure of the donor site, in turn reducing its morbidity.[1] When an expander is placed in a subcutaneous pocket, the pattern of blood supply to the overlying skin is altered. Myocutaneous perforating vessels are divided, and as a result, the skin flap may initially show signs of ischemia. However, as expansion proceeds, there is an increase in the number and size of the vessels within flaps supplied by random-pattern vessels and, if present, axial vessels.[2] These changes correspond to the demonstrated increase in blood flow to expanded flaps.[3] The blood supply of the flap also may be enhanced by the vascular network within the capsule which forms around the expander.[4]

For an expanded flap to survive when isolated on its axial vessels, the pedicle must be capable of perfusing the expanded periphery of the flap without becoming dependent on the observed increase in random vasculature. The present study demonstrates survival in a series of island flaps raised after tissue expansion and compares this with survival in a series of control flaps raised without prior expansion. The study also demonstrates any changes that might occur in the territory of the axial vessels as a result of expansion.

Materials and Methods

The pig buttock flap was chosen as a suitable model.[5] It is a large flap which can be raised as an island flap on the deep circumflex iliac artery (Fig. 1). When raised acutely, the flap has a surviving length of approximately 13 cm in animals of the size used in this study.

Ink injection studies and dissections were first performed on carcasses. Branches of the deep circumflex iliac artery were found to perfuse the flap, although the main arterial trunk coursed across the anterior border of the flap to run down the anterior surface of the hind leg, supplying the overlying skin (Fig. 2). The artery therefore had to be divided where it crossed the inferior border of the flap when the flap was raised as an island.

Six Yuccatan Grottinger miniature pigs with a mean weight of 20 kg were used in the study. Each animal was premedicated with azaperone and then anesthetized with 2% halothane, 69% oxygen, and 29% nitrous oxide, breathing spontaneously through a mask. On each side of the animal a buttock flap was marked out. In the first animal the flaps were 10 cm in breadth and 17 cm long. There was almost complete survival

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This paper was presented at the Summer Meeting of the British Association of Plastic Surgeons, in Liverpool, England, on July 10, 1986.

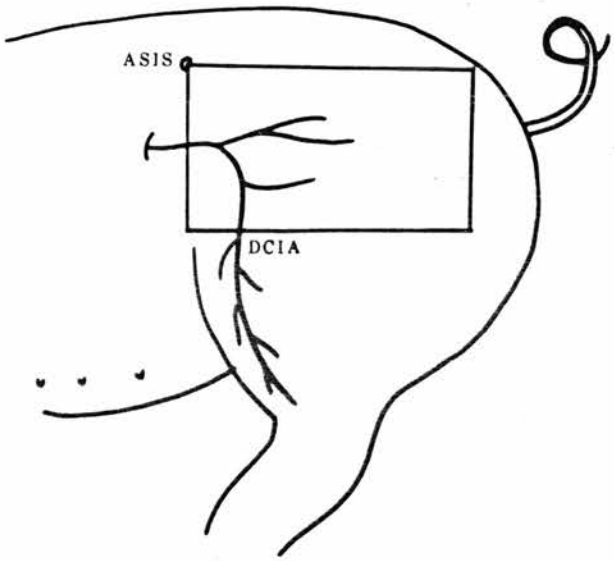

FIG. 1. Diagram of pig buttock flap. This flap has its cephalodorsal corner over the anterosuperior iliac spine (*ASIS*) and can be raised as an island on the deep circumflex iliac artery (*DCIA*).

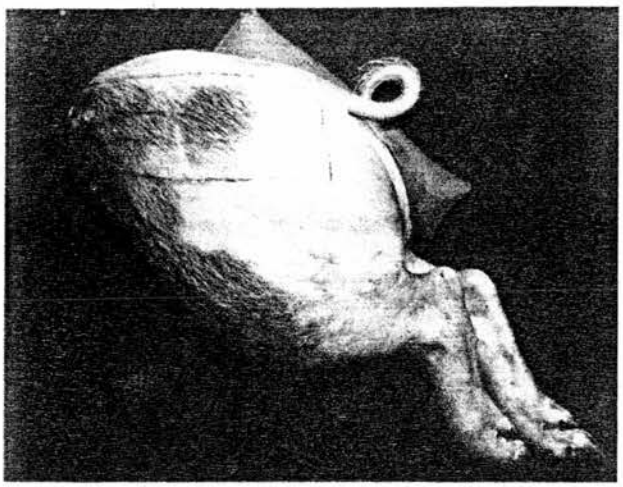

FIG. 2. Indian ink injection study of the deep circumflex iliac artery in a pig carcass. Perfusion of the buttock flap is demonstrated, although the main territory of the artery is seen to be the skin over the anterior surface of the hind leg.

of this pair of flaps, and so in the remainder of the animals the length was increased to 20 cm, the breadth remaining 10 cm An incision was made along the dorsal margin of the flap, and the flap was completely undermined by dissection with scissors. A 15 × 8 cm rectangular tissue expander* was placed under each flap, and the wounds were closed with an absorbable monofilament suture. The outline of the flaps was tattooed into the skin with carbon colloid. No intravenous fluids were given, no drain was used, but perioperative penicillin was administered.

At weekly intervals under sedation with intramuscular azaperone the expander on one side only of each animal was inflated until it felt firm, the other side being left empty as a control. In the first pig, a total of 750 ml was injected, but in order to provide a greater challenge to the flaps, in the remaining animals 1000 ml was used. This latter volume produced an increase of between 1.5 and 2 times the area of the control flap depending on the compliance of the skin (Fig. 3). Since the flap breadth was not a critical factor in flap survival, the length only was studied (a slight variation in breadth would have introduced a large element of error into comparisons of flap area).

When expansion was complete, between 6 and 9 weeks later, each animal was reanesthetized and both flaps were raised and isolated solely on their vascular pedicles (Fig. 4). The flaps were then sutured back in their beds with both expanders left in situ, without alteration in volume, to prevent flap shrinkage. Since the expanded flap was by this stage nearly hemispherical in cross section, it would have not been possible to prevent shrinkage by simply extending the donor defect to accommodate the increased area of the expanded flap. In addition, the presence of the expanders prevented any beneficial effect which may have resulted from an increase in the vascularity of the beds from affecting flap survival.

One week later the flaps were examined and areas of obvious necrosis were recorded. Measurement was made of the surviving and total lengths at each edge and in the midline of the flaps, and from these results the mean surviving and mean total lengths of each flap were calcu-

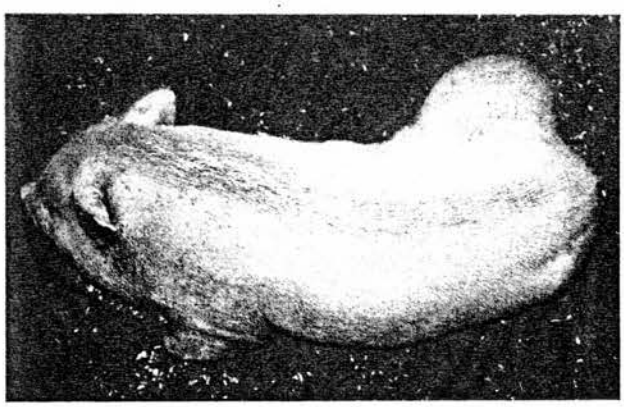

FIG. 3. Pig with one buttock flap expanded with 1000 ml saline.

* Cox Uphoff International, 2740 So. Harbour Blvd. Santa Ana, Calif.

lated. Using the paired *t* test, flap survival of the expanded and the control flaps was compared using the mean surviving lengths and then using the percentage of flap survival in each group.

After sacrificing the pigs with intravenous barbiturate, angiograms were performed on the axial vessels by injecting barium sulfate aqueous suspension at constant pressure. Changes that occurred in the vascular architecture were demonstrated.

FIG. 4. Pig showing buttock flap raised as an island on the deep circumflex iliac artery. Vascular pedicle arrowed.

FIG. 5. Pig at completion of experiment showing relative amounts of necrosis on expanded and control sides.

TABLE I
Surviving and Total Flap Lengths (in cm)

Pig	Expanded Flap		Control Flap	
	Surviving	(Total)	Surviving	(Total)
1	25.5	(26.5)	18.5	(19.0)
2	31.0	(34.0)	23.0	(24.0)
3	29.0	(31.0)	20.0	(22.5)
4	36.0	(39.0)	23.0	(24.0)
5	31.0	(31.0)	23.0	(23.0)
6	32.0	(32.0)	23.0	(23.0)
Mean	30.8	(32.3)	21.8	(22.6)

RESULTS

The mean weight of the animals was 26.5 kg at the end of the study. This increase in weight was due to growth, which also accounted for the increase in size of the control flaps.

In most animals there was some necrosis in both the expanded and control flaps (Fig. 5). The mean surviving and total flap lengths for each pig are shown in Table I. The mean of the surviving lengths of the expanded flaps was 30.8 cm and that of the control flaps was 21.8 cm. This demonstrates an increase in surviving length of nearly 50 percent following expansion, and this increase was found to be significant when tested (paired *t* test; $p < 0.001$). The surviving flap length measured as a percentage of total flap length is shown in Table II, with a mean of 95.7 percent of the expanded flaps surviving and 96.7 percent of the control flaps, which was not significantly different.

DISCUSSION

The insertion of a tissue expander, even if left empty, will enhance survival of an island flap. This has already been shown in studies of random-pattern flaps and occurs because the initial undermining of skin causes an effective delay of

TABLE II
Surviving Flap Length Measured as a Percentage of Total Flap Length

Pig	Expanded Flap	Control Flap
1	97%	99%
2	91%	96%
3	94%	89%
4	92%	96%
5	100%	100%
6	100%	100%
Mean	95.7%	96.7%

the flap.[6] This study shows that a flap of much greater size can be produced by tissue expansion prior to raising and islanding the flap. In this study, surviving lengths after expansion were approximately 50 percent greater than the delayed controls and nearly 150 percent greater than comparable flaps raised acutely.

Angiograms of the flaps not only demonstrated the changes previously shown in random-pattern flaps,[2] but also showed a considerable increase in the caliber of the axial vessel of the expanded flap compared with that of the control flap (Figs. 6 and 7). It is intended to make quantitative measurements of changes in blood flow to the expanded tissue in further experiments. The increase in vessel size has implications in the

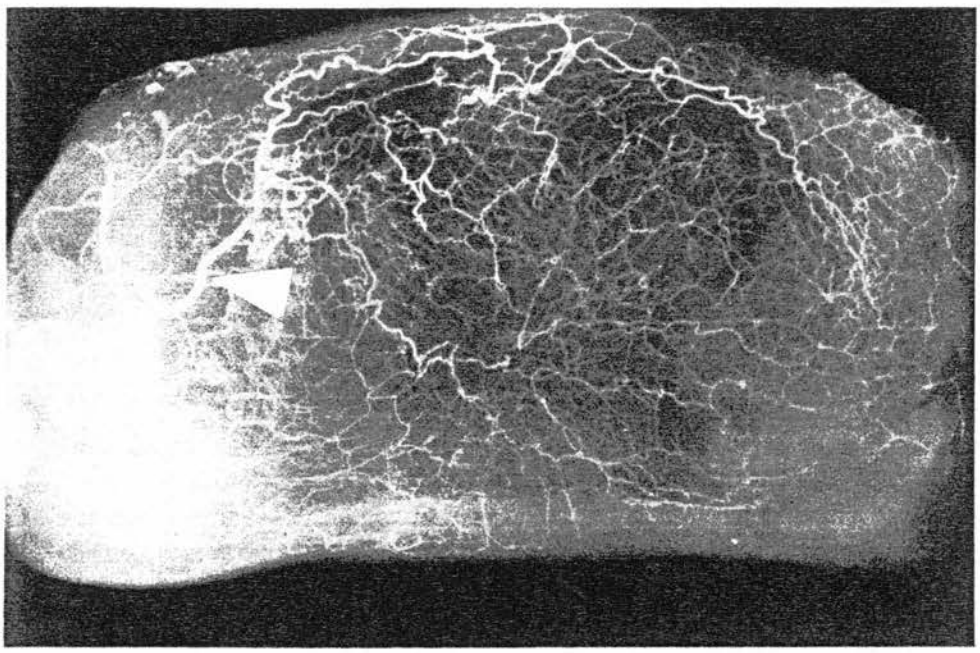

FIG. 6. Microangiogram of axial vessel of expanded flap demonstrating an increase in caliber of the vessel compared to that of the control flap from the same animal (seen in Fig. 7). Main vessel is arrowed.

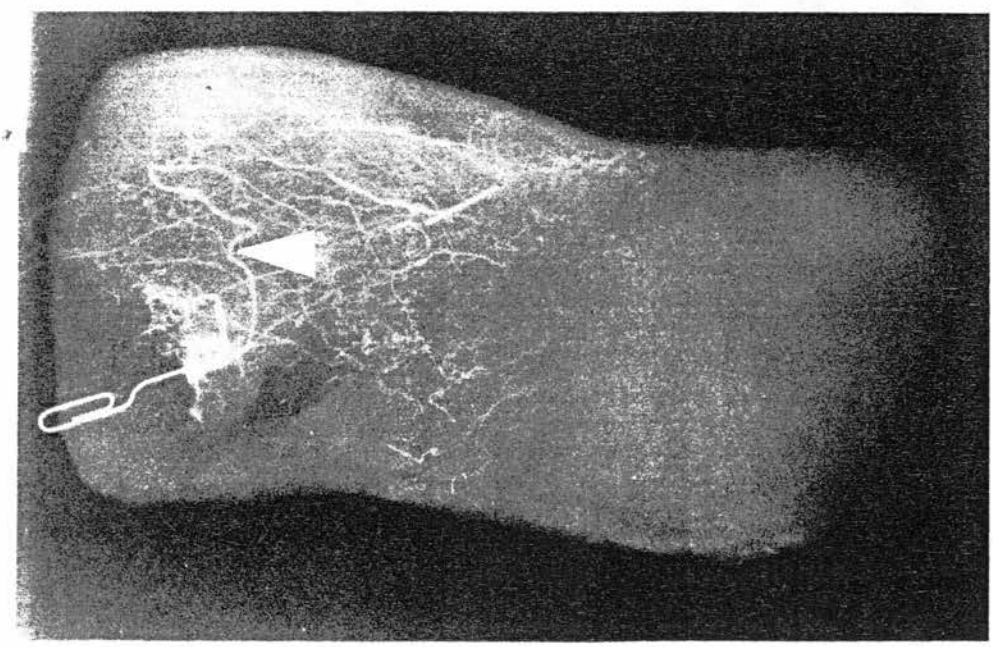

FIG. 7. Microangiogram of axial vessel of control flap. Main vessel is arrowed.

free tissue transfer of such flaps. A larger-diameter vessel would provide an easier and more reliable anastomosis. The capsule also may be of use in tissue transfer because its presence enables the flap to be raised quickly on removal of the tissue expander and it also provides a supportive and well-vascularized but nonbleeding backing.

Preexpansion of an island or free flap is a technique which can increase the size of a flap without jeopardizing its reliability.

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ACKNOWLEDGMENTS

I wish to thank Cox Uphoff International for providing the tissue expanders and Ethicon Ltd. for providing the P.D.S. sutures used in the project. In addition, I wish to thank Dr. George Cherry and Mr. Michael Masser for advice given during the study.

This work was funded by a grant from the Wessex Regional Health Authority.

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